THE PLANT DISEASE REPORTER

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PLANT DISEASE EPIDEMICS
and
IDENTIFICATION SECTION

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The Plant Disease Reporter is issued as a service to plant pathologists throughout the United States. It contains reports, summaries, observations, and comments submitted voluntarily by qualified observers. These reports often are in the form of suggestions, queries, and opinions, frequently purely tentative, offered for consideration or discussion rather than as matters of established fact. In accepting and publishing this material the Plant Disease Epidemics and Identification Section serves merely as an informational clearing house. It does not assume responsibility for the subject matter.

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PLANT DISEASE EPIDEMICS AND IDENTIFICATION SECTION

Horticultural Crops Research Branch — Plant Industry Station, Beltsville, Maryland

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TOBACCO BLUE MOLD EPIPHYTOTICS IN THE FIELD

Paul E. Waggoner and G. S. Taylor

In Connecticut, blue mold of tobacco caused by Peronospora tabacina is most destructive in seedbeds but frequently also causes important losses of tobacco in shade tents and open fields (1, 2). Losses from a heavy shower of Peronospora spores are likely to be large after several light disease years when the growers tend to omit control measures (7). To inform growers of impending danger, a blue mold warning service was initiated in Connecticut following a severe epiphytotic in 1951.

Warnings are issued on the basis of 1) the presence of primary infection in the tobacco growing region, 2) weather observations, and 3) the spread of the pathogen in an inoculated, experimental field of tobacco. The experimental field is located near New Haven; this is within the same climatic region as the commercial tobacco growing region but at a safe distance from it.

No warnings were issued in 1952 or 1953. No more than isolated occurrences of blue mold were reported. However, warnings were issued in June, 1954. Considerable blue mold occurred, particularly where thorough preventative measures were not taken. At the same time the pathogen was spreading from the source plants onto other plants in the experimental shade field. This spread, as indicated by the appearance of blue mold lesions, was mapped in order that it could be compared with the spread of other pathogens (4, 5, 8). In addition, the spread of the pathogen in a field of commercial, open-grown Broadleaf tobacco was mapped.

Spread in Shade Tobacco

The field of Connecticut 49 shade tobacco consisted of 16 north-south rows 3 feet apart and 66 feet long under a tent of shade cloth (2). The plants were set at 14-inch intervals on May 28. On June 11, day 0, the plants had about six expanded, succulent leaves. The plant at the center of row 8 and the one at the center of row 9 were inoculated with Peronospora collected from diseased seedlings.

On day 5 at 4 p.m., five yellow lesions without sporulation were apparent on the inoculated plants. By 10 a.m. on day 6 23 lesions were producing spores on the two plants. The weather conditions during this period are given in Table 1. Temperature and humidity observations were made under the tent; wind observations were taken by the Weather Bureau at the New Haven Airport. Wind directions at the airport and at the experimental field would be the same at night. The significant observation is that north and east winds prevailed in the evening and early morning during the period of spore appearance. Temperatures were low, humidity was high, and the soil was still wet from rains on the preceding days. The temperature in the tent during the following day, day 7, remained below 80° F. However, the temperature on days 8 through 11 was above 80° for 7 to 8 hours daily.

On day 13 "dry weather" lesions of blue mold (3) were evident throughout the southwest quadrant of the field where spores had probably been carried on days 5 and 6. The distribution of these lesions is shown in Figure 1. Only two other lesions were found outside this quadrant; one was 15 plants north of the source, another was 16 plants north and two rows east of the source. On day 17 sporulating lesions were observed on six plants outside the source. However, dry weather prevented these spores from producing new infections.

A gradient of spore concentration existed about the source plants during primary spread. It was estimated about an idealized source midway between the two actual source plants. The number of lesions per leaf was the basic observation. The leaves were 6 to 8 inches in length on day 13. Severely diseased leaves had 60 lesions per leaf. Evidently the number of lesions had an upper limit of about 100 although the number of spores that could land on the leaf was much greater. The possibility of multiple hits producing a single lesion was corrected for by Thompson's transformation (4). The transformed numbers were assumed to be the numbers of spores. These were then divided by the areas of the corresponding 3 feet wide annular rings around the source. The resulting gradient of spore concentration is shown in Figure 2.

The map and gradient, Figures 1 and 2, represent primary infection from a source that was approximately a point. They are comparable to the observations of the distribution of primary infection around sources of Phytophthora infestans, the late blight pathogen (5, 8). Peronospora spread about twice as far and produced two to three times as many lesions as Phytophthora did in a comparable late blight epiphytotic (8). Had the soil surface been covered with tobacco plants as it was with potatoes there probably would have been even more blue mold.

The gradients of blue mold and late blight infection can also be compared by means of a hypothesis concerning spore dispersal, the hypothesis being based on the statistical theory of the turbulent transfer of matter by air (4). Gradients of disease are described by this hypothesis in terms of source strength and of proportion of spores settling out per unit area passed over. Distribution of primary late blight infection has been described by means of this hypothesis. The proportion of Phytophthora spores settling out was estimated to be 0.15 (8). When the blue mold observations were subjected to the same analysis, the estimate of proportion of Peronospora spores settling out was about the same. The two estimates of source strength were not in the same units and could not be compared.

The farther spread of Peronospora as compared to Phytophthora could have been caused by one of several factors. First, it could have been due to a narrower, longer shape of the area of distribution. The maps of distribution show that this is not the case; both distributions were restricted to a single quadrant. Second, the greater spread could have been caused by a lower rate of settling out of the spores in the tobacco than in the potatoes owing to the smaller size of the tobacco plants. However, the analysis of the observations in terms of the turbulent transfer hypothesis argues against this reasoning.

Finally, the greater spread could have been due to a greater "source strength" increasing infection at all distances. The greater number of blue mold than of late blight lesions indicates that this is the case. Several factors affecting apparent source strength can be evaluated. First, there were not more blue mold than potato blight lesions in the source; in fact, there were fewer. Second, the weather was more favorable for both sporulation and infection in the case of the blue mold than in the case of the late blight epiphytotic. This undoubtedly increased both the number of spores per source lesion and the number of infections per air-borne spore. Third, relative to the number of spores produced in the source, more Peronospora than Phytophthora spores could have become air-borne owing to the mechanism by which the former are shot into the air (6). Evidently the second and third factors were more important than the first.

The conclusion drawn from these observations on a blue mold epiphytotic in shade tobacco is that a few lesions can lead to a primary infection for a distance of 15 yards downwind and very little upwind. Beyond this distance few lesions will be found.

Spread in Open Field Tobacco

Blue mold does occur in open fields of tobacco although it is more prevalent in shade grown tobacco (1). A detailed study of an epiphytotic in an open, commercial field of Broadleaf tobacco in 1954 reveals the manner of spread of the pathogen.

The location of the seedbeds, three tobacco fields totalling about 5 acres, and adjacent potato fields and woods is shown in Figure 3. The map is drawn to a scale of 20 yards per inch.

Plants were transplanted from the beds shown in Figure 3 to north-south rows in the fields between June 5 and 16. Blue mold was first noticed in the beds on June 22. The infection had become severe enough by July 2 that the beds smelled of decomposing plants and were destroyed. Infection was noticed in the field on July 1 and became severe by July 9, 17 days after the first infection was noticed in the beds.

The infection in the field was mapped on July 12-13 (Figure 3). The plants had eight to ten expanded leaves. The top three leaves were not yet infected. The number of lesions on every third plant was counted in 11 rows in field A and in 3 rows each in fields B and C. The number of lesions per plant decreased from over 200 near the seedbeds to as low as 0 in the far fields. However, most of the plants, even in the far fields, had at least one lesion. In the wet, shady northeast corner of field C were numerous lesions. Two other foci of infection were also observed.

The disease gradient is evident in Figure 3. The three foci of infection in field C probably resulted from setting infected plants in a location favoring the disease. However, the seedbeds were the chief source of infection. Had the beds been continuously protected and destroyed earlier, there would have been much less damage in the adjacent field.

A field of shade tobacco lay about 250 feet east of field C. About 4 percent of the plants in this field were infected, but nearest to field C, 4 to 20 percent of the plants were infected. This contrasts with 80 percent of the plants infected in field C. The dispersal of spores from the Broadleaf to shade is possible because, as our experiments have shown, the spores can pass freely through the shade cloth.

A field of Broadleaf tobacco about 200 feet southwest of field A had 4 percent of the plants infected in the corner nearest field A. Only about 1 percent of the plants in the remainder of the field had lesions.

Table 1. Weather observations on June 16 and 17[a].

Date and hour	Temperature	Relative humidity	Wind direction and speed	Sun
16 4 p.m.	66°F.	80%	E 18 mph	5 min.
5	66	73	E 17	40
6	66	65	E 12	0
7	60	70	E 7	
8	56	90	NE 6	
9	53	>95	NE 6	
10	52		NE 6	
11	50		NE 8	
12	49		NE 6	
17 1 a.m.	49		N 6	
2	48		N 8	
3	48		N 8	
4	49		N 8	
5	49		N 9	0
6	51	>95	N 8	60
7	55	85	NE 8	
8	60	80	E 9	
9	64	66	SE 11	
10	68	63	SE 12	
11	69	55	SE 13	
12	70	52	SE 15	
1 p.m.	71	60	SE 12	
2	71	65	E 12	
3	70	66	SE 13	
4	69	70	E 13	60
5	67	68	E 12	50
6	63	70	E 8	0
7	60	85	E 7	
8	56	>95	NE 5	
9	54		NE 5	
10	52		NE 5	
11	50		N 3	
12	48	>95	N 2	0

[a] The observations were taken at the experimental field except the wind observations which were taken by the Weather Bureau, New Haven Airport.

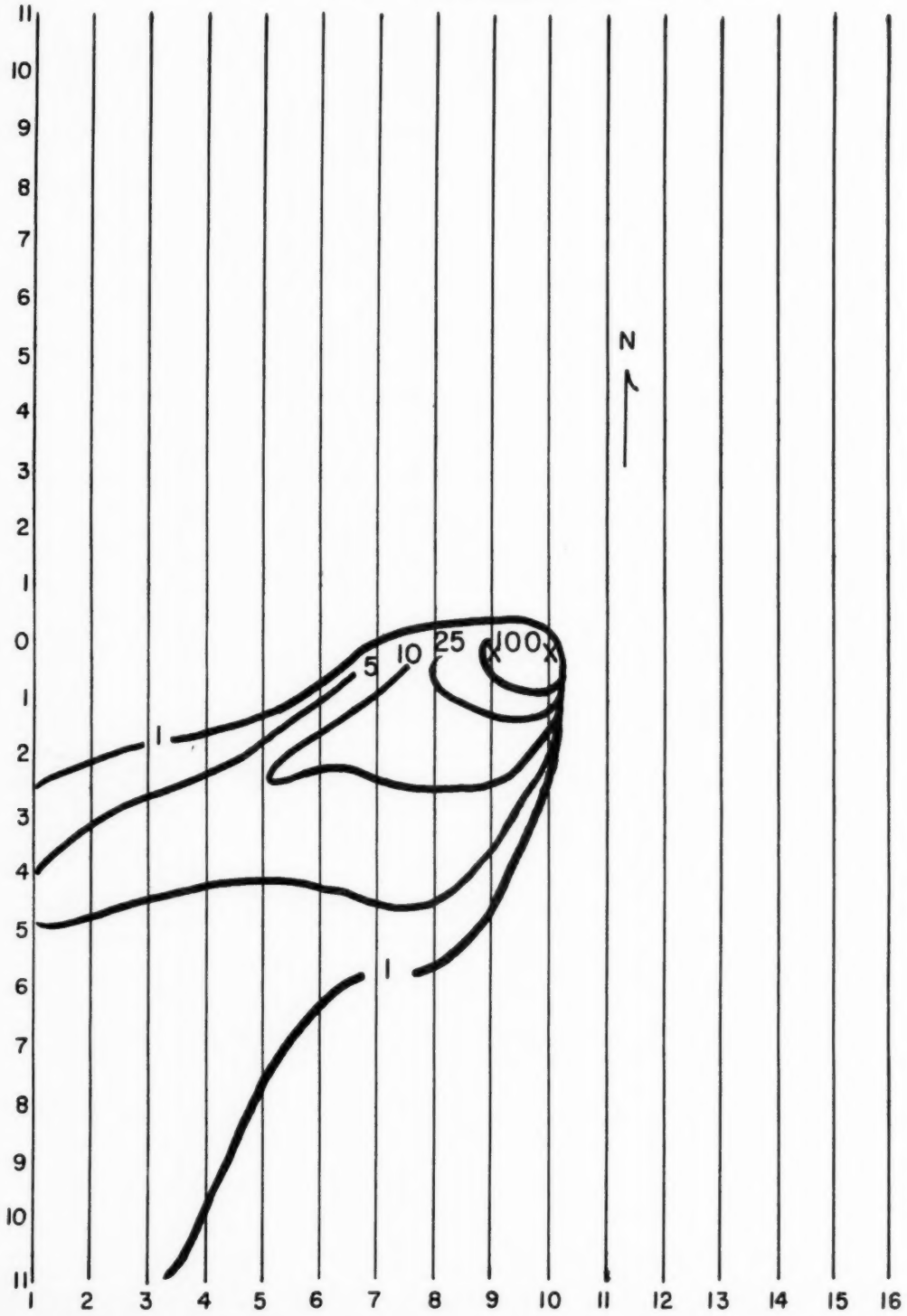

FIGURE 1. Distribution of number of blue mold lesions per plant around a source of Peronospora tabacina (X) in shade grown Connecticut 49 tobacco.

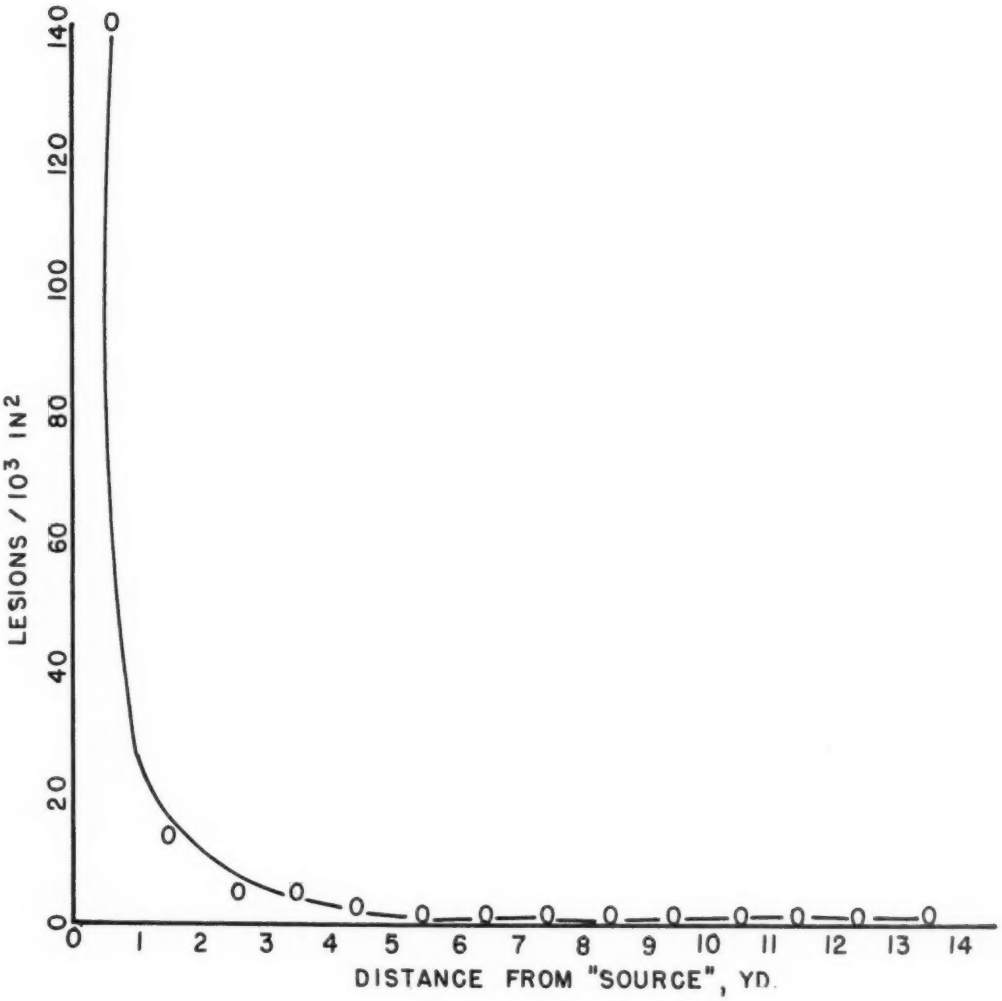

FIGURE 2. Number of blue mold lesions per unit area of field at distances from the source of Peronospora tabacina.

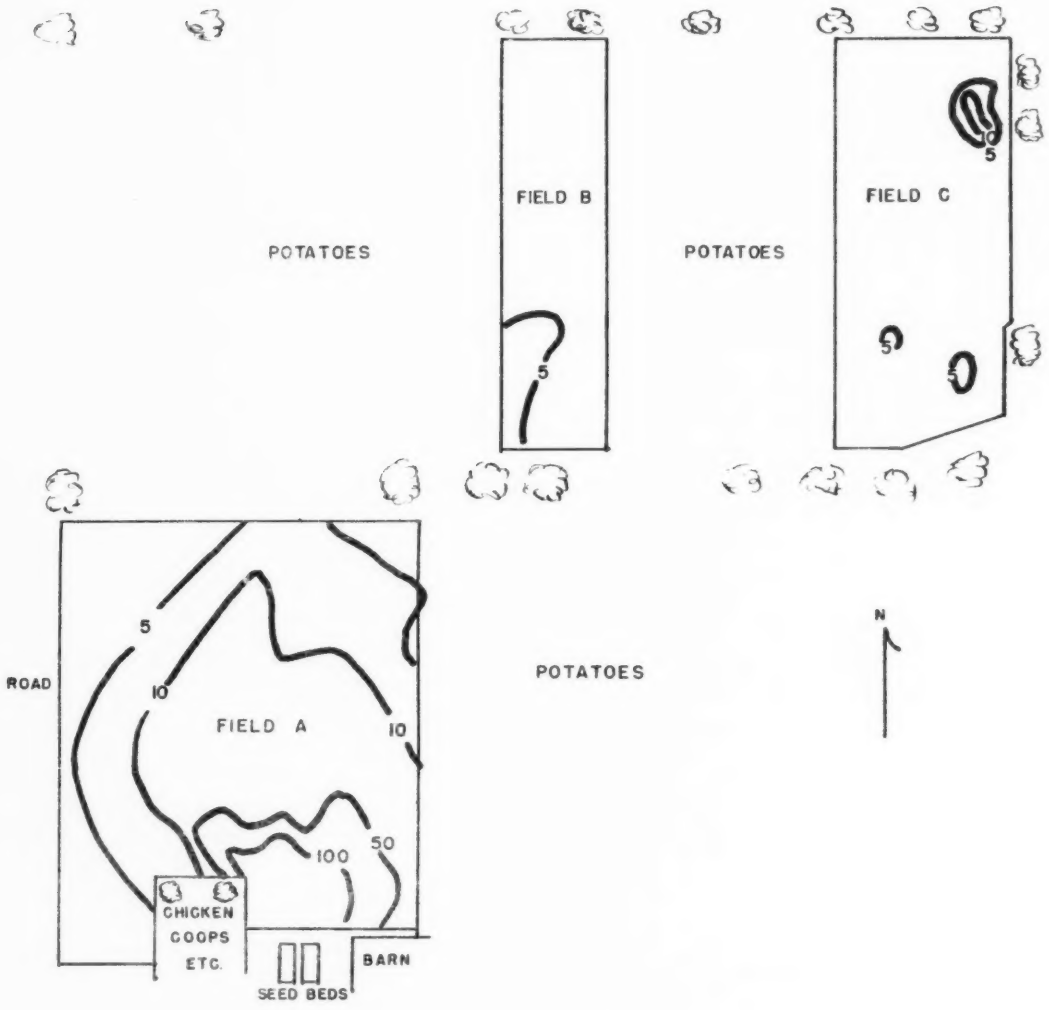

FIGURE 3. Distribution of number of blue mold lesions per plant in open grown Broadleaf tobacco. One inch represents 20 yards.

The observations that a blue mold epiphytotic involved a restricted area of open field tobacco is consistent with the preceding observation that an epiphytotic involved a restricted area of shade grown tobacco.

Conclusion

Peronospora tabacina spores from blue mold lesions lead to primary infection for a distance of several yards downwind and very little upwind. Beyond this distance few lesions will be found. Therefore, sources of infection on his own farm are the primary danger to a grower's tobacco. Infections produced in a distant or even a neighboring field are much less numerous. Therefore, each grower will realize large benefits from sanitation on his own farm. This is particularly true of seedbeds, which should be thoroughly protected against blue mold infection up to the time they are destroyed. This is to prevent them from becoming sources of field infection. The seedbeds should then be destroyed as soon as possible.

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CONNECTICUT AGRICULTURAL EXPERIMENT STATION, NEW HAVEN

A SURVEY OF LATE BLIGHT OF POTATOES IN AROOSTOOK COUNTY, MAINE AUGUST 26 -- SEPTEMBER 10, 1954

Reiner Bonde

A quick survey in Aroostook County showed that 2.4 percent of the potato fields were completely dead from late blight (Phytophthora infestans); 39.9 percent were not attacked; 56.9 percent had less than 5 percent infection, and 68 percent less than 10 percent. No attempt was made to estimate the loss in acres or barrels of potatoes.

The survey was made between August 26 and September 10. Only fields bordering automobile highways were examined because of the time element involved. Only fields of an acre or larger are included in the survey figures.

The surveyors, namely E. S. Schultz, Senior Pathologist of the United States Department of Agriculture, Forrest Welch, Field Assistant, Clement Dunning, Assistant County Agent, and Reiner Bonde, Plant Pathologist of the Maine Agricultural Experiment Station, were not able to examine all fields critically. Nevertheless, the data obtained from the rapid survey are sufficiently accurate to give a true picture of the situation, which is not so bad as it was first thought to be. The loss caused by late blight should not be minimized, however, because of possible subsequent losses in storage.

Generally, it has been a poor year in Aroostook County. Weather records show 18 rainy days during May, 15 in June, 22 in July, and 19 in August. Such conditions resulted in late planting, washouts, and flooded fields, and often made it impossible to get into the fields with heavy control machinery. In addition, the weather was very favorable for blight throughout the season. Practically all of the growers used very large amounts of fungicides in an attempt to curb the blight but weather conditions contributed greatly to minimizing the effects of the treatments. The picture is not entirely black, however. Large growers with good equipment were able to take full advantage of what good weather there was, and in most cases obtained good blight control; better in fact than they had expected.

One circumstance of particular interest is that many of the blight-free fields were planted to potatoes of the Kennebec variety.

Table 1. Extent of potato late blight infection in Aroostook County, Maine. Surveyed August 26 to September 10, 1954.

	Southern Aroostook	Central Aroostook	Northern Aroostook	Total
Number of fields examined	487	1139	534	2160
	Percent of total fields in each area			
No blight	35.7	40.8	41.6	39.9
Trace up to 5	20.1	15.2	18.0	17.0
5 to 10	10.4	10.8	12.4	11.1
10 to 25	7.4	7.5	8.6	7.7
25 to 50	4.9	5.5	7.3	5.8
50 to 75	4.9	4.8	4.1	4.7
75 to 90	5.5	4.8	2.8	4.4
90 to 95	3.7	4.1	3.2	3.8
95 to 98	3.9	3.9	0.9	3.2
100	3.5	2.6	1.1	2.4
Total	100.0	100.0	100.0	100.0

The survey covered 487 potato fields in the southern part of Aroostook County, 1139 fields in the central section, and 534 fields in northern Aroostook; a total of 2160 potato fields of an acre or more in area.

Table 2. Percentage of late blight infection in Aroostook County, by towns. Surveyed August 26 to September 10, 1954.

Towns	Number of fields	Percentage of fields surveyed showing			
		None to 10% blight	10 to 50%	50 to 90%	90 to 100%
Northern Aroostook:					
Cyr	43	48.8	32.7	11.6	6.9
Fort Kent	129	75.9	10.0	7.8	6.3
Frenchville	32	59.5	21.8	12.5	6.2
Limestone	108	91.7	4.7	0.0	3.6
Madawaska	47	65.9	21.4	10.6	2.1
New Sweden	43	65.3	20.9	6.9	6.9
Notre Dame	49	49.0	26.5	16.4	8.1
Van Buren	83	77.1	16.8	2.5	3.6
Central Aroostook:					
Ashland	69	53.5	7.3	20.4	18.8
Blaine	45	75.5	13.4	0.0	11.1
Bridgewater	74	64.8	16.2	12.2	6.8
Caribou	234	78.6	6.1	6.8	8.5
Castle Hill	16	43.6	25.2	12.5	18.7
Easton	61	83.6	8.3	3.2	4.9
Fort Fairfield	235	63.0	15.3	10.2	11.5
Mapleton	58	62.1	10.3	8.6	19.0
Mars Hill	81	45.7	23.4	16.1	14.8
Presque Isle	172	68.1	17.4	9.3	5.2
Washburn	40	47.5	15.0	15.0	22.5
Westfield	54	81.5	9.4	3.6	5.5
Southern Aroostook:					
Dyer Brook	26	76.9	7.6	7.8	7.7
Island Falls	26	69.2	19.3	11.5	0.0
Houlton	58	82.7	6.9	8.7	1.7
Limerick	24	62.5	4.2	12.5	20.8
Linneus	23	43.4	21.8	13.2	21.6
Littleton	71	66.2	11.3	11.2	11.3
Monticello	139	59.0	19.4	10.8	10.8
Oakfield	33	27.3	12.1	18.1	42.5
Patten	13	77.0	0.0	7.6	15.4
Sherman	37	91.9	5.4	2.7	0.0
Smyrna	17	58.8	11.8	17.6	11.8
Staceyville	20	95.0	0.0	5.0	0.0

In southern Aroostook the Limerick, Linneus, and Oakfield areas were hard hit, with 12.5, 13, and 15.2 percent respectively of the fields surveyed being in the 100 percent blight class and showing no green foliage. In each of these areas the small number of fields surveyed allowed purely local conditions to influence the percentages.

On the credit side of the ledger for southern Aroostook, large numbers of fields surveyed in Dyer Brook, Houlton, Littleton, Monticello, Sherman, and Staceyville showed little blight. Of that group the largest percentage of 100 percent blight fields showed up in Monticello, where 3.6 percent of the 139 fields surveyed were showing no green foliage. In contrast, 36 percent of the fields surveyed in Monticello were very green with practically no blight showing.

Central Aroostook, with 1139 fields in the survey, showed only two areas with more than 5 percent of the fields in the 100 percent blight class. Of the 74 fields in Bridgewater, 5.4 percent were completely blighted. Of the 40 fields in Washburn, an even 5 percent were dead.

Caribou, with 234 fields surveyed, had 47.4 percent in the group showing little or no blight. Fort Fairfield with 235 fields showed 34.9 percent of them with little or no blight, and in Presque Isle with 172 fields in the survey 38.4 percent of them were placed in the little or no blight class.

Easton with 61 fields had 59.0 percent of them in the little or no blight class.

Northern Aroostook with 534 fields in the survey seemed to have the least blight damage with only 1.1 percent of its fields in the 100 percent blight class, and 41.6 percent of them showing little or no blight. The greatest losses were in the Cyr and New Sweden areas where 2.3 percent of the surveyed fields showed no green foliage.

At the other extreme, Limestone with 108 fields surveyed had 84.2 percent in the little or no blight class. In the northern part of the County a greater percentage of its fields showed 5 to 10 percent blight than in the southern or central areas. The difference, however, was not great.

Since the unfavorable weather continued up until the beginning of the harvesting season, many farmers had little or no opportunity to do an effective job of top-killing. Growers were warned, therefore, that all possible precautions should be taken to make sure that the blight spores were not carried into the tubers to cause storage rot, which could add materially to the already severe loss throughout the County. Information on the development of storage rot will be gathered at a later date.

Tables 1 and 2 give the complete survey results in the three sections of Aroostook.

MAINE AGRICULTURAL EXPERIMENT STATION

FUNGICIDES FOR DOWNY MILDEW CONTROL ON HEADING CABBAGE

William M. Epps

Cabbage downy mildew (Peronospora parasitica Fr.) regularly causes moderate to heavy losses in the South Carolina Coastal area. It first appears on the lower leaves of the fall crop cabbage in September, several weeks before the plants begin to head. As the heads develop, the disease spreads from the lower leaves to the wrapper leaves and to the exposed leaves on the top. Often systemic infection results in indefinite necrotic areas on the midribs and leaf blades in the lower portion of the head.

Downy mildew affects the market value of the crop in several ways. The lesions on the head and wrapper leaves detract from appearance. They may also serve as routes of entry for secondary pathogens, such as soft rot bacteria, which may cause decay in transit. The presence of numerous lesions on the top of the head also appears to render that portion of the head more susceptible to frost injury.

The spring crop, which is planted in seedbeds in early October, is often attacked as soon as the young plants emerge. The plants are killed or stunted, depending on the severity of the attack. Damage to the heads of the spring crop is only rarely sufficiently severe to warrant the use of fungicides for mildew control. Serious damage occurred in the spring crop in 1954 for the first time in several years.

Table 1. A comparison of fungicidal dusts for the control of downy mildew on heading cabbage. Charleston, South Carolina 1950-1953.

Fungicide[a]	Concentration (% active)	Percent of heads severely diseased 1950	1951	1952	1953
Control	-	43.7	33.8	32.2	10.4
Spergon (chloranil)	5.0	3.1	6.0	4.4	0.6
Spergon (chloranil)	10.0	--	4.2	6.1	0.0
Dithane Z-78 (zineb)	3.9	21.2	4.	29.1	--
Phygon XL (dichlone)	1.0	14.7	--	--	--
Copper No. 30	4.0	--	--	16.6	--
Thiram	5.0	--	--	16.7	--
Manzate (maneb)	4.2	--	--	18.0	--
Vancide F995W	6.0	--	--	22.3	--
Vancide 51ZW	6.0	--	--	22.5	--
Ethyl B-622	4.0	--	--	--	3.2
Pennsalt 1282	--	--	--	--	7.4
Minimum difference required for signficance at the 5% point		8.7	7.6	10.0	4.8

[a Chemical names of active ingredients: Spergon -- tetrachloro-p-benzoquinone. Dithane Z-78 -- zinc ethylenebis (dithiocarbamate). Phygon XL -- 2, 3-Dichloro-1, 4, naphthoquinone. Thiram -- bis(Dimethylcarbamoyl) disulfide. Manzate -- manganous ethylenebis(dithiocarbamate). Vancide F995W -- manganese dimethyldithiocarbamate; 2-mercaptobenzothiazole. Vancide 51ZW -- zinc dimethyldithiocarbamate; zinc 2-mercaptobenzothiazole. Ethyl B-622 -- 2, 4-Dichloro-6-(o-chloroanilino)-sym-triazine.]

Foster (6, 7), reported that chloranil (Spergon and Dow Seed Protectant No. 5) was the most effective fungicide tested for the control of downy mildew in cabbage seedbeds in Mississippi. Eddins (1, 2, 3, 4, 5) in Florida tested a wide variety of fungicides, both on young plants in the seedbed and on heading plants in the field. He concluded that chloranil (Spergon) was the most effective of all the materials tested in both seedbed and field. He recommended initially the use

of nabam spray in the field, however, even though it was less effective than chloranil dust or spray, because of the high cost of the chloranil. His early tests were conducted with a dust containing 12% chloranil. His most recent recommendations (5) call for use of chloranil spray, nabam spray, 6.5% zineb dust, or 5% chloranil dust.

Tests were initiated in South Carolina in 1950 and repeated annually for a period of four years to see if effective control of downy mildew on heading cabbage could be obtained and if less expensive dust schedules could be developed. Dusts were applied to the heading cabbage at 5-day intervals beginning about 2 weeks before the first harvest and continuing throughout the harvest period. Six applications were adequate, except in 1953 when the development of the crop was delayed by cool dry weather and eight applications were necessary. Dusts were applied with a Root Hand duster with a single nozzle per row directed straight down onto the head.

Since almost all heads showed some disease, the degree of control obtained could only be determined by grading the heads into classes based on severity of infection. The heads were separated into three classes as follows: 1) healthy -- no lesions on the head or wrapper leaves; 2) light to moderate symptoms -- lesions present but not sufficiently numerous to coalesce and result in tissue breakdown; 3) severe symptoms -- lesions numerous and coalescing so as to result in breakdown of tissue of the outer leaves. Only the percentages of severely diseased heads are presented in Table 1. These figures appear to offer the best and most efficient means of comparing the effectiveness of the various chemicals.

Chloranil (Spergon) gave the most effective control of downy mildew of all the fungicides tested. Some of the other chemicals, notably thiram, Manzate (maneb), Copper No. 30, Ethyl B-622, and Phygon (dichlone), effected a statistically significant reduction in the percentage of severely diseased heads, but all of these fungicides, with the exception of B-622, were significantly inferior to chloranil. The dust containing 5% chloranil proved equal to the 10% dust except in 1953. This difference, not shown in the Table, resulted in a higher percentage of healthy and lightly diseased heads (75.5 percent) where the 10% dust was used than in the plots which received 5% dust (62 percent). This reduction in the concentration of chloranil from 10% or 12% to 5% tends to bring the cost of the chloranil dust down to where its field use is economically feasible.

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CLEMSON COLLEGE TRUCK EXPERIMENT STATION, CHARLESTON, SOUTH CAROLINA

A PROGRESS REPORT ON GRAY MOLD AND GHOST SPOT OF TOMATOES AND THEIR CONTROL[1]

J. F. Darby[2]

Summary

Gray mold and ghost spot have become serious diseases of tomatoes in Florida. The symptoms of both diseases are described as they occur in the field.

Results of experiments in the greenhouse to reproduce ghost spot were partly successful. Histological studies failed to show that ghost spot was caused by an insect or a fungus; however, the nature of the injury suggested the latter.

In fungicide trials conducted over a three-year period (1951-54), 50% wettable dichlone (3/4: 100) was the most effective material tested for the control of both gray mold and ghost spot; however, it was ineffective against gray leaf spot. This difficulty was largely overcome by alternating dichlone with zineb, nabam-zinc sulfate, or maneb. Mixtures of dichlone and zineb (1/2: 1 1/2: 100; 3/8: 1: 100) were found to be promising in the control of gray mold, ghost spot, and gray leaf spot.

INTRODUCTION

Gray mold of tomatoes, caused by the fungus Botrytis cinerea Fr., has been known in Florida for many years. Heretofore, it has been considered only as a minor fruit rot, occurring mostly in transit. In recent years it has become recognized as a serious disease in the field, where it attacks the foliage as well as the fruit. Spraying with nabam and zinc sulfate, zineb or maneb has not controlled it. Only a few years ago, the disease in the field appeared to be largely restricted, geographically, to the Indian River area. Since that time, the diseased area has been gradually extended, so that at present gray mold poses a serious problem throughout most of southern Florida. Losses attributed to gray mold have ranged up to 50 percent of the yield.

The occurrence and location of a disease known as ghost spot have closely paralleled that of gray mold. Both diseases first became serious in the Indian River area just after World War II, and the areas now severely affected by the diseases are approximately the same. Losses due to ghost spot are difficult to estimate because they depend in part on prevailing market conditions. When the demand for tomatoes is good, fruits showing a few ghost spots are either ignored or placed in a lower grade, while only the severely spotted fruits are rejected; however, when the demand is poor all spotted fruits are rejected.

In 1938 Ainsworth, Oyler, and Read[3] presented evidence to show that a spotting of tomatoes (indistinguishable from ghost spot in appearance) was caused by B. cinerea. To account for the spotting, they suggested the following explanation: Under conditions of high humidity, Botrytis spores lying on the surface of immature fruits germinated and penetrated the epidermis, forming the enzyme pectinase in the process. These sporelings later died with the return of drier conditions and no fungus could be isolated; however, a halo was formed by the enzyme and a necrotic speck marked the point of partial entry of the organism.

Field observations have usually supported the theory that ghost spot is caused by B. cinerea. With few exceptions, conditions favorable for gray mold resulted in a typical rot of the fruit, whereas unfavorable conditions resulted in ghost spot. Occasionally they occurred on the same fruit.

This paper presents information concerning symptoms of gray mold and ghost spot in the field and the results of experiments designed to determine the relationships between the two. A summary of the results of the fungicide treatments that were effective in controlling gray mold and ghost spot is also presented.

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[1]Florida Agricultural Experiment Station Journal Series, No. 369.

[2]Formerly Assistant Plant Pathologist at Indian River Field Laboratory, Ft. Pierce, now located at Central Florida Experiment Station, Sanford, Florida.

[3]Ainsworth, G. C., Enid Oyler, and W. H. Read. 1938. Observations on the spotting of tomato fruits by Botrytis cinerea Pers. Ann. Appl. Biol. 25: 308-321.

GRAY MOLD

Botrytis cinerea ordinarily does not attack growing tomato tissue directly, but invades it after first becoming established on dead or weakened tissue. Once established, under favorable weather conditions, it attacks actively growing foliage, stems, and fruit. Gray mold has usually appeared in late fall and remained a threat until early spring. The plants seem to be most susceptible when they are approaching maturity. Large, rank vines have been the most severely damaged.

The first easily recognizable sign of the disease is the occurrence of a dense gray mold which develops on the dead leaves and stems underneath the vines (see Fig. 1). This mold consists of conidiophores with large numbers of conidia which are easily dislodged and become wind-borne. Many of them finally come to rest on nearby healthy tomato plants which, in turn, become diseased under conditions of favorable moisture and temperature.

The first symptom of infection is a watersoaking and softening of the affected tissues. This is followed by the appearance of mycelium and spores. The whole fruit is sometimes affected (Fig. 2). The fungus enters the leaves through any injured area, such as that caused by leaf miners and diseases other than that caused by B. cinerea. After the affected leaf tissues collapse, the fungus enters the stem and produces characteristic cankers (see Fig. 3). Fruit may become infected when diseased leaves come in constant contact with the fruit (Fig.4). From this area of contact the fungus may enter the fruit and produce a soft watery rot which may extend over the entire fruit. Initial penetration also occurs through growth cracks and scars at the stem and stylar ends. Entry may also occur through injuries such as those caused by sand and fingernails.

GHOST SPOT

Ghost spot has been serious only in the spring. It has appeared mostly on young fruit ranging in size from 1/2 to 1 1/2 inches in diameter. With few exceptions ghost spot has been found only in fields where gray mold had occurred earlier in the season.

Ghost spot appears on the surface of green tomatoes as small, whitish rings or halos mostly ranging in size from 3 to 6 mm. in diameter. The color of the areas within the circles formed by these rings is a lighter shade of green than the outside. There is usually a very small brown necrotic speck at or near the center of each ghost spot. This speck is slightly raised above the surface of the fruit and does not extend any deeper into the fruit than the epidermis. As the fruit matures and turns red, the color of the rings changes from whitish to gold. There are several variations in the rings, as is shown in Figure 5. Usually there is a single ring with a rather indefinite border, but some appear to be a solid whitish disc, while others have rings within rings, each with a definite border. These spots have not been known to increase in size or number during storage. They apparently do not affect the quality of the fruit other than to mar its appearance.

When ghost spot first becomes evident in the field, the spots are most numerous on fruit located on the more exposed portions of the plant. Fruits from the inner, more protected areas either lack ghost spots or have them on the shoulder and necrotic specks without the halo on the blossom end. Ghost spots predominate on the shoulder of the fruit; whereas true gray mold commonly occurs on the blossom end.

EXPERIMENTAL RESULTS IN THE LABORATORY AND GREENHOUSE

Attempts to isolate an organism from ghost spots were unsuccessful. Preliminary attempts to reproduce ghost spots with Botrytis conidia using Ainsworth's[3] method resulted in the production of necrotic specks similar to those found in the center of ghost spots, but no haloes were formed.

Histological studies were conducted in an effort to determine the nature of the necrotic spots in the center of the ghost spots. Paraffin sections, varying in thickness from 8 to 20 microns, were stained in safranin and counterstained in fast green or cotton blue. There was no puncture in any of the necrotic spots examined which might suggest penetration by an insect. No evidence of an infection peg or mycelium was found in or near the necrotic tissue. A layer of elongated cells or wound tissue surrounded the necrotic areas. The necrotic areas were deeply stained with safranin, suggesting lignified or cutinized cell walls or tissue of basic character.

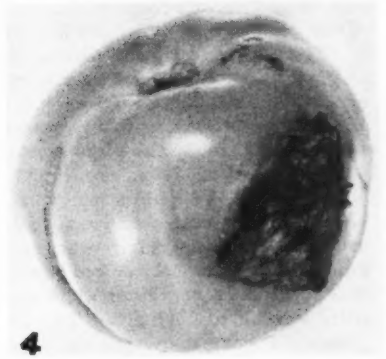

FIGURE 1-4. Gray mold. 1 -- Gray mold on foliage, stems, and fruit underneath a heavily infected tomato vine. 2 -- A tomato fruit rotted by gray mold. The fuzzy growth at the left is composed of spore masses of the fungus. 3 -- Canker formation following gray mold infection. Note elliptical shape, light tan color and concentric rings. The black fungus on the right canker is a saprophytic species of Alternaria. 4 -- A tomato fruit with a leaflet infected with gray mold pressed against it. The fungus has entered the fruit from the leaflet.

FIGURE 5. Ghost spot. Variation in types of ghost spots on tomato fruits of various sizes.

METHODS IN THE FIELD

All of the materials employed in these experiments were in the form of a spray. (Active ingredients of the chemicals are: dichlone -- 2, 3-Dichloro-1, 4-naphthoquinone, nabam -- Disodium ethylenebis (dithiocarbamate); Zineb -- Zinc ethylenebis (dithiocarbamate); maneb -- Manganous ethylenebis (dithiocarbamate); PCNB -- pentachloronitrobenzene). In tests conducted in the winters of 1951-52 and 1952-53, the applications were made with a small, self-propelled power sprayer adjusted to deliver 250 to 300 p.p.s.i. at the pump. This sprayer was equipped with a hand-carried boom and a shut-off valve. The boom was attached to a 100-foot hose which was pulled between the rows to reach various parts of the field. The sprayer used in the winter of 1953-54 was a high clearance (6 feet), tractor-mounted, independently-powered sprayer adjusted to deliver 300 p.p.s.i. at the pump. From 3 to 12 nozzles per row were used, depending upon the size of the plants.

Intervals between applications varied from 3 to 7 days, depending upon severity of the diseases. The average number of applications per season was around 20. The gallons per acre applied ranged from 50 to 250 in proportion to the size of the plant.

Four replicates, arranged in a pattern of random distribution, were used for each treatment. The spacing between plants within rows was 2 feet and the inter-row distance was 7 feet. Plots were separated by single guard rows to minimize the effect of the drifting of materials. Each longitudinal half of the guard rows was sprayed with the material applied to the adjacent plots. Thus, there was an overlapping of spray materials on the guard rows. In the winter of 1951-52, the plots consisted of 2 rows, each of which were 28 feet long. In the following winter, 1952-53, the length of these rows was extended to 30 feet. In the winter of 1953-54, each of the 4 plots per treatment was enlarged to 8 rows 80 feet long. In the latter test the data were collected from the 2 rows in the center of the 8-row plots.

Dichlone-zineb mixtures were prepared by thoroughly mixing proportionate quantities of the wettable powders of dichlone and zineb together. These mixtures were stored in closed, circular cardboard containers and used as needed. No evidence of deterioration or incompatibility was noted during this period.

The plots were inspected daily for the presence of diseases. When there appeared to be enough differences between treatments to warrant taking data, all plants were examined and scored.

The tomatoes were picked mature-green, weighed, and counted at approximately weekly intervals. Each fruit was examined at the time of harvest for the presence of lesions or rots. The fruits were sorted into two classes only: marketable and unmarketable.

Insecticides were included in the tank with the fungicides once a week. The insecticides used were wettable parathion or DDT or a mixture of the two.

RESULTS AND DISCUSSION OF FIELD TRIALS

In earlier trials using small, standard-sized plots dichlone decreased the incidence of gray mold, but it was not so effective as the other fungicides in the control of gray leaf spot (Stemphylium solani Weber) (Table 1). There was also an indication that it might be effective against ghost spot. Observations on disease control in earlier trials on the guard rows where there was an overlapping of spray materials indicated that a mixture of dichlone and zineb might be promising in the control of both gray leaf spot and gray mold. In a fall trial in 1953, a dichlone-zineb mixture (1/2: 1 1/2: 100) was included to check this possibility. This mixture was more effective than dichlone against gray leaf spot, and the two fungicides were compatible with each other and with wettable DDT and/or parathion. As usual in early fall crops, gray mold and ghost spot were not present, therefore its effectiveness against those diseases was not determined.

Since Botrytis spores are produced in such huge quantities and are easily wind-borne, they could be disseminated over the small standard-sized plots from adjacent heavily infected plots, thereby reducing the apparent effectiveness of a fungicide. In the hope of obtaining more accurate information on gray mold, a trial was conducted in the winter of 1953-54 using large, 8-row plots. Each of the 8 rows was sprayed, but data were taken from the 2 center rows. The data from this large-plot trial are presented in Tables 2 and 3. It should be noted that the incidence of gray mold and ghost spot was quite low; this should be considered in interpreting the data. As is presented in Table 2, dichlone, dichlone-zineb mixture, and dichlone alternated with other materials reduced the amount of gray mold and ghost spot below that of zineb-treated

Table 1. The relative effectiveness of a selected group of fungicide formulations on the control of gray mold, ghost spot, and gray leaf spot of tomatoes in small plots (0.036-0.038 acre per treatment).

Material and dosage	Percent fruit showing			Incidence of gray leaf spot[a]	
	Gray mold		Ghost spot		
	1951-52	1952-53	1952-53	1951-52	1952-53
Dichlone 3/4: 100	0.00	0.97	0.50	8.17	23.50
Nabam-$ZnSO_4$ 2 qts.: 3/4 lb.: 100	10.80	10.41	1.33	2.15	0.50
Zineb 2: 100	1.30	5.07	2.12	4.42	1.50
Maneb 1 1/2: 100	11.30	8.55	1.91	4.27	0.75
No fungicide	0.00	0.92	2.23	34.85	57.87
L.S.D. @ 5% for 9 treatments	3.20			9.58	
13 treatments		2.28	N.S.D.		13.04

[a]The figures for 1951-52 represent the average number of gray leaf spot lesions per leaf, while those for 1952-53 are the estimated percentage defoliation due to gray leaf spot.

Table 2. The relative effectiveness of various fungicide formulations on the control of gray mold and ghost spot of tomatoes in large plots (0.41 acre per treatment) when the infection levels remained low.

Material and dosage	Gray mold			Ghost spot	
	Percent plants showing	Foliage index[a]	Percent fruit harvested	Percent fruit harvested	Percent fruit[b]
Dichlone 3/4: 100	0.00	0.00	0.03	0.03	15.00
Zineb 2: 100	75.58	0.83	0.70	1.49	55.00
D-Z mixture 1 3/8: 100[c]	0.00	0.00	0.25	0.21	23.00
D-Z-N-M-C alternate[d]	18.30	0.28	0.35	0.30	37.00
Zineb-PCNB alternate 2:100[e]	43.30	0.43	0.31	1.00	40.00
No fungicide	0.00	0.00	0.00	0.76	5.00
L.S.D. @ 5%	26.30	0.39	0.31	0.95	15.95

[a]Figures are based on the following ratings: 0 = minimum; 5 = maximum.
[b]These data were collected 1 week after the last harvest and 12 days after the last fungicide application. The fruits selected were from 1 to 1 1/2 inches in diameter.
[c]Wettable powders of D (dichlone) and Z (zineb) were mixed at the ratio of 3/8:1, respectively.
[d]N (nabam) zinc sulfate 2 qts. : 3/4 lb. : 100; M (maneb) 1 1/2: 100; C (Copozim) 6: 100. The alternate schedule of applications was: Z-Z-M-M-C-N-D-D-N-D-M-N-M-D-D-M-D-D-D-Z-Z-D.
[e]PCNB (75% wettable pentachloronitrobenzene). The schedule of applications was: Z-Z-Z-Z-Z-Z-PCNB-PCNB-Z-Z-Z-Z-PCNB-Z-PCNB-Z-Z-PCNB-Z-Z-Z

Table 3. The relative effectiveness of various fungicide formulations on the control of gray leaf spot, the size of the vine, and yield of tomatoes in large plots (0.41 acre per treatment) when the infection levels remained low.

Material and dosage	Gray leaf spot severity[a]	Density and size of vine[a]	Yield in marketable bu./acre
Dichlone 3/4: 100	1.37	4.50	147.20
Zineb 2: 100	0.56	4.19	139.03
D-Z mixture 1 3/8: 100[c]	1.00	4.25	142.16
D-Z-N-M-C alternated[d]	0.81	4.38	156.11
Zineb-PCNB alternate[e]	1.62	3.81	130.11
No fungicide	3.62	3.12	43.23
L.S.D. @ 5%	0.71	0.52	34.58

See Table 2 for footnotes.

plots. The dichlone-zineb mixture and dichlone alternated with other materials also gave a better control of gray leaf spot than did dichlone alone (Table 3).

It has been noted that where defoliation or a reduction in density and size of tomato plants was found, there was a corresponding reduction in the severity of gray mold. Thus, the unsprayed check plots and the plots sprayed with materials ineffective against the leaf-spotting diseases usually had the least amount of gray mold. This was thought to be because defoliation permitted more air and sunshine to penetrate and dry out these plants, making conditions unfavorable for the development of gray mold. The data presented in Table 3 were collected to show whether the control of gray mold was due to defoliation or to the fungicide. These data show no correlation between treatments containing dichlone and a reduction in the density and size of plants. Neither do they show any reduction in yield. They do show that the control of gray mold and ghost spot obtained by the zineb-PCNB alternate and the unsprayed check plot could be due to the failure of these treatments to control the leaf spotting diseases.

The data and observations to date strongly suggest that ghost spot is caused by Botrytis infections that failed to become sufficiently established to cause a rot. Such a relationship is assumed until further research throws more light on the subject.

CENTRAL FLORIDA EXPERIMENT STATION, SANFORD, FLORIDA

SUSCEPTIBILITY OF ALLIUM SPP TO PUCCINIA ASPARAGI[1]

Louis Beraha[2]

Summary

Experiments have demonstrated that several species of Allium and all the horticultural varieties of A. cepa tested were susceptible to P. asparagi. Uredia developed on all the horticultural varieties of A. cepa tested, and were followed by telia in most cases. No reaction differences were detected among the various species of Allium that would aid in determining races of P. asparagi.

Although Allium cepa has been reported as susceptible to Puccinia asparagi DC. only the pycnial and aecial stages have been found on it in nature (5, 8). Sheldon (6) in 1903 reported successful infection resulting in production of all four stages of the fungus on onion, but these results have not been repeated. The lack of further inoculation work, coupled with the failure to find uredia or telia on varieties of Allium cepa even when growing in fields adjacent to infected asparagus plants, made it desirable to investigate further the susceptibility of Allium cepa as well as other species of Allium to infection by P. asparagi. If the various species of Allium differed in type of reaction to rust infection, they might serve as index plants to detect the existence of races of P. asparagi. At present Asparagus officinalis L., A. scaber Baker, A. maritimus Pall., and A. acutifolius L. are of no value for this purpose, since all these species show a "very susceptible" reaction type (2), analogous to type 4 of the widely used cereal rust scale.

In the experiments described in this paper, the susceptibility to P. asparagi was determined for seven species of Allium, for the progeny of two inter-specific crosses, and for eight horticultural varieties of Allium cepa.

The method of inoculation was essentially the same as that employed for the cereal rusts (7). Urediospores were collected by tapping heavily-rusted asparagus seedlings lightly over a porcelain dish and were diluted with talc at the rate of 1 part urediospores to 20 parts talc. Leaves of onion seedlings to be inoculated were atomized lightly with distilled water, rubbed between moistened fingers to remove the bloom, and dusted with the urediospore-talc mixture. Following a 24-hour period in an inoculation chamber held at 100 percent relative humidity and 60° F., the plants were returned to a well-lighted section of the greenhouse maintained at 75° to 85° F.

Results

Preliminary attempts to inoculate 3- to 4-week-old seedlings of Allium cepa were unsuccessful. Subsequent trials were made using single plants in the six- to eight-leaf stage, while the multiplier-type onions were at least 2 months old. Infection was obtained on Allium fistulosum (Welsh or Spanish onion); A. ascalonicum (shallot onion); A. schoenoprasum (chives) as well as progeny of crosses involving A. cepa X A. fistulosum (open-pollinated), and A. ascalonicum X A. fistulosum (amphidiploid)[3]. In addition, uredia, followed in most instances by telia, developed on all of the following horticultural varieties of Allium cepa: Brigham Yellow Globe, Large Red Wethersfield, Southport Red Globe, Crystal White Wax, Golden Globe, Bunching, White Portugal, and Sweet Spanish (Riverside strain). Other species of Allium, viz., A. porrum (leek), A. sativum (garlic) and A. ampeloprasum (wild leek), seemed to be immune as no infection resulted on either seedlings or mature plants.

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[1]This report is a summary of a portion of a thesis presented to the Graduate School of the University of Illinois in 1954 in partial fulfillment of the requirements for the degree of Doctor of Philosophy. The writer expresses his appreciation to Dr. H. W. Anderson under whose direction the work was done.

[2]Pathologist, Fruit and Nut Crops, Agricultural Research Service, Horticultural Crops Research Branch, Beltsville, Maryland.

[3]Species and crosses of Allium were kindly furnished by Dr. August E. Kehr of the Department of Horticulture at Louisiana State University.

The first indication of infection on onions was the development of an oval chlorotic patch varying in size from a few millimeters to 1 cm. in length and 3-5 mm. in width. These patches appeared at maturity to be composed of three distinct zones. The first zone consisted of a necrotic center within which sparsely-sporulating uredia appeared after 14 to 17 days. The second zone, immediately adjacent, was pale green, and was often surrounded by a narrow chlorotic band that formed the third zone. Sporulation on any of the varieties or species was sparse and the symptom complex resembled the 0;-1 type infection assigned to rust-resistant varieties of cereals (Fig. 1). At times, however, spores could be found in elevated sori on the underside of the leaf (within the leaf's lacuna or cylinder). Curiously enough sporulation on A. schoenoprasum was observed only on the upper surface of the leaf. The chlorotic patches on the onions were associated with a progressive wilting and drying of the tips, particularly of the extremely susceptible outer (oldest) leaves. This correlation between age of leaf and susceptibility was especially pronounced. Newly-formed leaves were immune while certain portions of somewhat older leaves exhibited discrete spots. However, these leaves in general did not show tip wilting. In no instance did the type of infection on any of the species of Allium or varieties of A. cepa differ enough to distinguish the response of any one suscept from that of any other.

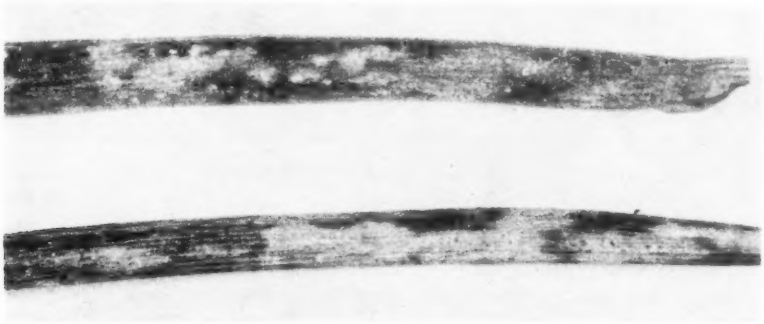

FIGURE 1. Uredial sori of P. asparagi on upper surface of leaves of Allium cepa, showing minute sori surrounded by oval chlorotic zones.

Urediospore inoculum from asparagus from three widely-separated areas in Illinois was increased on asparagus in the greenhouse. Spores from these sources caused infection of all the susceptible onions but did not give different types of infection.

Since the uredial and telial stages of P. asparagi have not been reported from natural collections on onions, it might be suggested that the infection obtained in onions was due to Puccinia porri (Sow.) Wint., the causal agent of the common rust of onions and garlic. However, the evidence is strongly against this possibility, for the urediospores from asparagus used in numerous trials did not infect garlic, while P. porri is reported to incite garlic rust (4); also urediospores removed from infected onions produced typical rust symptoms on asparagus when used in inoculations, while attempts made to infect asparagus in California with a mass urediospore collection of P. porri from rusted garlic plants were unsuccessful (9). In addition, morphological features of both urediospores and teliospores produced on onion agreed with descriptions given by Arthur for P. asparagi (1) and confirmed by Dr. G. B. Cummins[4].

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FRUIT AND NUT CROPS, AGRICULTURAL RESEARCH SERVICE, HORTICULTURAL CROPS RESEARCH BRANCH, BELTSVILLE, MARYLAND

EFFECT OF IRRIGATION ON BEAN ROOT ROT[1]

H. J. Walters[2]

Root rot of beans is a perennial problem to growers where beans are grown under irrigation. The disease may be caused by several species of fungi, but the Fusarium group is responsible for most of the rot in Wyoming. These organisms attack the taproot and usually cause a reddish discoloration which may extend to the soil line. The main root and lower part of the stem may become dry and pithy. The rootlets that develop from the taproot are killed. When this occurs, a cluster of rootlets develops above the lesion immediately below the soil line and keeps the plant alive.

Since root-rotting organisms are normal inhabitants of practically all soils, the best that can be done is to make conditions as favorable as possible for the bean plant so that it will thrive in spite of the parasites in the soil. The fact that the roots of severely infected plants are near the soil line presents a problem of keeping sufficient moisture in that zone. If conditions are made favorable for these infected plants, a nearly normal crop may be produced.

Table 1. Pounds of beans produced per acre with three varieties of beans and two irrigation practices. Experiments in Wyoming, from 1950 through 1953.

Variety	Irrigation practice[b]	Yield (pounds/acre)				Average over 4 years
		1950	1951	1952	1953	
Great Northern UI 16[a]	Light-Frequent	1811	----	1499	1585	1656
	Heavy-Infrequent	1529	----	1371	1404	1435
Great Northern UI 123	Light-Frequent	1814	2075	1618	1680	1797
	Heavy-Infrequent	1630	1966	1380	1276	1538
Pinto UI 78	Light-Frequent	2087[c]	2428[c]	1856	2451	2211
	Heavy-Infrequent	1582	2213	1656	2251	1926

[a]UI refers to varieties developed by the University of Idaho.
[b]See text.
[c]Significant at the 5% level.

An experiment involving two irrigation practices was designed to ascertain whether frequency of irrigation and amount of water applied at each irrigation affect development of root rot and resulting yield of three varieties of beans. The two irrigation practices were: 1) Light-frequent: -- soil kept moist by frequent light irrigations, with first one made approximately 3 weeks after planting followed by a light irrigation at 5- to 7-day intervals throughout the growing season; and 2) Heavy-infrequent: -- soil allowed to become very dry and beans to acquire a dark green color before first irrigation, which was usually 6 weeks after planting, followed by two or three rather heavy irrigations when plants become dark green. The latter practice is the one used by most of the bean growers.

These experiments were conducted over a period of 4 years. Incidence of root rot was ascertained by selecting at random ten plants from each of the outside rows of four-row plots of each of four replications and indexing the severity of rot on the roots of each plant. Yields were taken from two center rows.

[1]Published with approval of the Director, Wyoming Agricultural Experiment Station, as Journal Paper No. 54.
[2]Assistant Plant Pathologist, Arkansas Agricultural Experiment Station, Fayetteville, Arkansas; formerly with Wyoming Agricultural Experiment Station, Laramie, Wyoming.

There appeared to be no correlation between the two irrigation practices and the severity of root rot. For the 4 years and three varieties of beans, yields were increased with light-frequent irrigations over heavy-infrequent irrigations (Table 1). The increases in yield were not always statistically significant at the 5 percent level, but were sufficiently high in the greater percentage of cases to be of economic importance.

Increased yields brought about by supplying water to the root zone of bean plants infected with root rot may be explained by the assumption that the moisture prevents wilting and makes nutrients available near the surface of the soil.

WYOMING AGRICULTURAL EXPERIMENT STATION, LARAMIE, WYOMING

CHEMICAL SOIL TREATMENTS FOR THE CONTROL OF RHIZOCTONIA ON SNAP BEANS

W. D. Moore and Robt. A. Conover[1]

The root rot disease of vegetable plants, caused principally by strains of Rhizoctonia solani Kuehn, has been of considerable importance on all soil types in Florida for many years (2), resulting in poor stands, weakened plants, and reduced yields. Research has indicated the advantages of seed treatments in some cases and the benefits of soil management practices in others, but no treatment has been devised thus far that will insure appreciable control under average field conditions. This is true particularly of snap beans.

Although seed treatments of snap beans have generally increased stands somewhat under Florida conditions, they have resulted in little or no reduction in numbers of Rhizoctonia stem lesions. This lack of control might be attributed either to weaknesses of the materials or to methods of application. Since the upward movement of the bean cotyledons through the soil at the time of germination removes a considerable amount of the chemicals from the root zone, the effectiveness of the treatments when applied to the seed is markedly limited. Although many chemicals have in the past shown varying degrees of effectiveness against Rhizoctonia, only within recent years have compounds become available that are highly specific for this fungus and at the same time not particularly phytotoxic. Arndt (3) found that pentachloronitrobenzene was highly effective against Rhizoctonia on cotton when applied over and around the seed in the soil, and Kendrick and Middleton (4) obtained similar results in both soil and pure culture tests with Rhizoctonia and other soil fungi. Brinkerhoff et al. (1) got effective results with pentachloronitrobenzene against Rhizoctonia in artificially inoculated rows, but failed to get control when the soil was wet and cold. In these cases, chemicals were applied over and around the seed at planting time or incorporated into the soil; thus a greater amount of material was used and better distribution than is provided by usual methods of seed treatment was obtained. The data reported in this paper are from field tests with snap beans in the mid-winter vegetable growing area of Florida in which chemicals were used in a manner somewhat similar to that reported by the workers cited.

Materials and Methods: Rows 25 to 100 feet long were used in these tests, with each treatment being replicated four times in a randomized design. Only chemicals that had controlled root rots on other crops or that had acted favorably against Rhizoctonia in laboratory tests were tested. Two types of application were used. First, bean seeds were dropped in open furrows and the required amounts of chemicals were applied as dusts or suspended in water and sprinkled over the seed and surrounding soil. The treated soil was then carefully pushed over the seed with a hand rake so that treated soil covered the seed. In the second method -- one readily adaptable to commercial use -- chemicals were applied as a spray through a nozzle attached at the rear of the shoe of a bean planter. The nozzle was adjusted so that the seed, the open furrow, and soil covering the seed were all covered by the spray. A hollow-cone nozzle was used and the spray was applied at the rate of 30 gallons per acre.

Tests were conducted during the winter growing season in south Florida on marl, muck, and sandy soils, which differ materially in pH, temperature, moisture, physical characteristics, and cultural requirements.

Bean plants were pulled when approximately 6 inches tall and the below-ground stem areas were examined for Rhizoctonia lesions. In some experiments only the percentages of plants infected were recorded. In other tests, plants were placed in four disease classes as follows: no lesions, light, moderate, and severe, the last class being characterized by girdling lesions or two or more long deep lesions exposing vascular elements of the stem. Since there was little difference between treatments in the light and the moderate disease classifications, only the data on severe infections are presented.

Results: Results of the first experiment comparing six chemicals are shown in Table 1. This was on marl soils with all treatments made by hand in open rows. It is apparent that the PCNB treatments were the only ones that reduced Rhizoctonia infection significantly and, also, that the heavier applications of this material were not significantly better than the lighter. Subsequently, several dosages of PCNB were compared with other seed or seedbed treating mate-

[1] Respectively, Senior Plant Pathologist, Agricultural Research Service, U. S. Department of Agriculture, and Plant Pathologist, Florida Agricultural Experiment Station

Table 1. Rhizoctonia infection of bean plants grown in marl soil following treatment with certain chemicals applied by hand at the time of planting.

Material	Rate per acre	Total plants examined	Percent infected with Rhizoctonia
PCNB 75% W.P.*	5 lbs.	413	13.4
" "	10 lbs.	365	7.9
" 20% dust	25 lbs.	406	9.1
" "	50 lbs.	379	6.9
Vancide 51	10 lbs.	404	54.3
Crag 5400	10 lbs.	381	45.7
Captan, 50% W.P.	10 lbs.	386	55.3
Dowicide A	2 lbs.	388	65.0
Dithane Z-78	10 lbs.	382	54.6
Check	-	400	57.9
L.S.D. @ 1%			21.5

*Pentachloronitrobenzene wettable powder.

Table 2. Rhizoctonia infection of bean plants grown in marl soil following treatment with certain chemicals applied as a spray at time of planting.

Material	Rate per acre (Active)	Total plants examined	Percent infected with Rhizoctonia
PCNB*	1 lb.	764	12.3
"	5 lbs.	660	2.9
"	10 lbs.	757	3.2
"	20 lbs.	864	2.4
Captan	5 lbs.	667	41.3
Thiram	5 lbs.	747	49.6
Chloranil	5 lbs.	616	34.6
Check	-	857	49.9
LSD @ 1%			20.8

*Pentachloronitrobenzene wettable powder.

rials on marl soil. These were applied as a spray during planting as described in the second method. Results from this experiment, shown in Table 2, again indicate the effectiveness of PCNB in reducing Rhizoctonia infection on beans. Although percentage infection decreased as dosage increased, there was no significant difference between the lowest and the highest rates of application of PCNB. Captan, thiram, and chloranil did not reduce Rhizoctonia infections.

Since PCNB was the most effective material used in the tests just described, additional dosage tests were made with the same amounts of PCNB on sand, muck, and marl soils. In addition, four dosages of material (No. 363) similar to PCNB were included in the experiments on sand and muck soils. Water suspension applications on the sand and on the muck soils were made by hand as described for the first experiment, whereas the treatments on the marl soil were applied as in the second experiment. Results from the tests on all soil types are shown in Table 3. It is apparent that PCNB reduced Rhizoctonia significantly on all soils, but it is of interest that the degree of control varied considerably with soil type, the variation being greatest on the marl soil and least on the muck. Although the experimental material No. 363 was tested in only two soils, the results suggest that it may be equal to PCNB in reducing Rhizoctonia infection on snap beans.

Table 3. Rhizoctonia infection on snap beans on three soil types following chemical treatment of seed and surrounding soil at time of planting.

Treatment lbs. active per acre	Sand: Total plants examined	Sand: Means of Rhizoctonia percentages	Muck: Total plants examined	Muck: Means of Rhizoctonia percentages	Marl: Total plants examined	Marl: Means of Rhizoctonia percentages
PCNB 1 lb.*	358	55.3	387	11.5	615	76.3
" 3 lbs.	319	31.1	356	18.7	557	37.2
" 5 lbs.	316	29.9	339	16.1	472	19.2
" 10 lbs.	352	13.0	369	4.2	601	13.4
363 1 lb.**	332	42.8	373	22.1	-	-
" 3 lbs.	362	20.6	326	18.2	-	-
" 5 lbs.	322	13.4	363	12.6	-	-
" 10 lbs.	355	11.6	381	9.1	-	-
Check	303	66.7	337	23.5	429	86.3
LSD @ 1%		23.5		11.0		9.1

*Pentachloronitrobenzene 75% W.P.
**Experimental material similar to pentachloronitrobenzene.

Discussion and Summary: Results presented in this paper indicate that both PCNB and the experimental compound No. 363 reduced Rhizoctonia infection on snap beans under field conditions. Since these materials are not water soluble and do not penetrate the soil readily, they require special methods of application for best results. Apparently, applications as sprays or dusts are effective if the materials are applied so that the seed and surrounding soil are well covered. Efficiency of the materials depends upon the thoroughness of their incorporation into the soil. In these tests considerable variation in degree of control occurred in different soil types when the same amounts of PCNB were used, the greatest response being found on marl soils and the least on muck. Nevertheless, in all cases significant reduction in disease incidence was obtained with amounts that would not be prohibitive in cost to the average grower.

In evaluating these results it should be noted that PCNB has little effect on other soil organisms which cause or are associated with damping-off and root rots under Florida conditions. In some instances it has seemed that obvious control of Rhizoctonia has not lessened the overall damage by root-rot. Also, the economic importance of Rhizoctonia infection on beans in Florida has not been determined; consequently the potential value of the effective treatments is not known. Although infections run above 90 percent in many fields and appreciable infection is found in all fields at all times, most growers produce average yields fairly consistently. The exceptions would be cases where Rhizoctonia so reduced the stands that the fields were abandoned or else replanted. Until such time as losses from this organism can be accurately determined, it appears that treatments for its control will have to be accepted for their potential rather than for their known value.

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AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE, AND FLORIDA AGRICULTURAL EXPERIMENT STATION COOPERATING

THE USE OF INSECTICIDES AS CORM AND SOIL TREATMENTS FOR CONTROL OF BACTERIAL SCAB OF GLADIOLUS

J. L. Forsberg

Abstract

The insecticides aldrin, lindane, and heptachlor applied to the soil at planting time effectively controlled bacterial scab of gladiolus. In soil which produced heavily scabbed corms when no treatment was used, the insecticides reduced scab about 90 percent. Application of the insecticides to the soil was more effective than was use of the same insecticides as corm treatments. The insecticides mixed with Arasan and applied to the corms dry were more effective than the same insecticides used in the emulsion form as wet treatments, either alone or combined with the fungicide, Emmi. Use of mixtures of Emmi and lindane and of Emmi and aldrin as corm treatments resulted in some injury. No injury resulted from use of mixtures of Emmi and heptachlor or from mixtures of Arasan and the insecticides.

INTRODUCTION

Control measures for bacterial scab of gladiolus, caused by Pseudomonas marginata (McCull.) Stapp, have consisted largely of pre-planting corm treatments with various mercury compounds. The possibility that the disease might be spread by soil insects was suggested by Young (3). He believed that the addition of an insecticide to the pre-planting corm treatment mixture might reduce the incidence of scab by killing or repelling soil insects capable of wounding the surface of corms and providing points of entry for P. marginata.

To test this possibility Young conducted experiments in 1951 and 1952, using mixtures of the fungicide New Improved Ceresan and the insecticides aldrin, heptachlor, and lindane. In these tests the incidence of scab on the corms grown from stock treated with either aldrin or lindane was considerably less than that on corms from stock treated with New Improved Ceresan alone or from untreated planting stock. With the best treatment, however, 17.6 percent of the harvested corms still carried scab lesions.

In a series of fungicide tests by the writer in 1953, DuPont I & D Seed Protectant was one of the materials included in trials for Fusarium rot control. When corms from one plot of the gladiolus variety Tivoli were cleaned it was found that corms produced from corms treated with I & D Seed Protectant were free from scab, whereas most of those from corms given other treatments were scabby. I & D Seed Protectant is a mixture of the fungicide, Arasan, and the insecticide, lindane.

In another test the same year the writer (1) found that aldrin placed under the corms in the planting furrow gave good control of white grubs (Phyllophaga spp.). Corms free from white grub damage were also free from scab, while many of the corms damaged by grubs also had scab lesions.

With the results obtained by Young and those obtained by the writer in 1953 in mind, a more extensive experiment was carried out in the growing season of 1954. Because of the good results obtained with the aldrin soil treatment in 1953 it seemed reasonable that if soil insects are instrumental in spreading the scab organism, better results might be obtained from soil treatments than could be obtained from corm treatments. Also, because of the apparent scab control obtained from use of I & D Seed Protectant it seemed desirable to compare the effects of wet and dry corm treatments.

MATERIALS AND METHODS

In comparative tests of various corm and soil treatments the following combinations were used:

Corm treatment	Soil treatment
1. Emmi, 30 ml. in 3 gallons; soak 15 minutes	None
2. Emmi, 30 ml. in 3 gallons; soak 2 hours	None
3. Arasan SF-X, dry	None
4. Mercuric chloride 1:1000; soak 2 hours	None
5. Aldrin emulsion, 23 ml. in 3 gallons; soak 15 minutes	None
6. Lindane emulsion, 28 ml. in 3 gallons; soak 15 minutes	None
7. Heptachlor emulsion, 23 ml. in 3 gallons; soak 15 minutes	None
8. Emmi, 30 ml.; aldrin, 23 ml. in 3 gallons; soak 15 minutes	None
9. Emmi, 30 ml.; lindane, 28 ml. in 3 gallons; soak 15 minutes	None
10. Emmi, 30 ml.; heptachlor, 23 ml. in 3 gallons; soak 15 minutes	None
11. Arasan SF-X, 2 parts plus aldrin 25% WP, 1 part, dry	None
12. Arasan SF-X, 4 parts plus heptachlor 50% WP, 1 part, dry	None
13. Arasan SF-X, plus lindane (DuPont I & D Seed Protectant)	None
14. Emmi, 30 ml. in 3 gallons; soak 2 hours	Aldrin
15. Emmi, 30 ml. in 3 gallons; soak 2 hours	Lindane
16. Emmi, 30 ml. in 3 gallons; soak 2 hours	Heptachlor
17. Arasan SF-X, dry	Aldrin
18. Arasan SF-X, dry	Lindane
19. Arasan SF-X, dry	Heptachlor
20. None	None

The aldrin emulsion contained 2 pounds aldrin compound 118 equivalent per gallon; the lindane emulsion contained 20% gamma benzene hexachloride; the heptachlor emulsion contained 2 pounds actual heptachlor per gallon. Du Pont I & D Seed Protectant contained 56% thiram from Arasan SF-X plus 14% gamma benzene hexachloride from lindane. Emmi is N-ethylmercuri-1, 2, 3, 6-tetrahydro-3, 6-endomethane-3, 4, 5, 6, 7, 7-hexachlorophthalimide.

Forms and amounts of insecticides used as soil treatments were as follows: aldrin, 20% granular, 5 grams per 10 feet of row; lindane, 25% wettable powder, 4 grams per 10 feet of row; heptachlor, 2 1/2% dust, 40 grams per 10 feet of row. In order to facilitate uniform application of the insecticides to the soil, the amount of chemical needed to treat 10 feet of row was mixed with enough dry soil to fill a 4-ounce jar. This soil-insecticide mixture was poured in the furrow immediately before the corms were set.

All corm treatments were made immediately before planting. No attempt was made to apply definite amounts of the dry materials to the corms. The corms and a small quantity of the chemicals were placed in a paper bag and shaken until the corms were thoroughly coated.

One hundred corms of each of the following planting stocks were used for each treatment: Lady Jane, size 2 and 3 corms, severely scabbed; Snow Princess, size 3 and 4 corms, mostly clean; Rosa Van Lima, number 3 culls, very rough, some scab and white grub damage; Rosa Van Lima, number 4, light scab; Spic and Span, number 3, severely scabbed; Margaret Fulton, number 3, very rough and scabby.

Each stock was planted on a different farm in the commercial gladiolus growing area in Kankakee County, Illinois. All plots were arranged in randomized block designs of 4 replicates with 25 corms per replicate.

RESULTS

Effects on Emergence -- Emergence counts made June 10, approximately one month after planting, are summarized in Table 1. A few plants emerged after this date but another count was not made.

Increased emergence resulted from some of the treatments in all plots except Spic and Span but these increases were not consistently associated with the same treatments. The outstanding result noted in the emergence counts was the poor emergence from corms given the Emmi plus lindane treatment. In four of the plots these counts were significantly lower than those obtained from untreated corms; in the other two plots they were essentially equal to those from untreated corms, and significantly less than counts from corms given other treatments. The Emmi-aldrin soak also appeared to reduce emergence counts in the Rosa Van Lima plots.

Table 1. Effects of corm and soil treatments on emergence of gladiolus plants.

Corm treatment	Soil treatment	Number of plants emerged from 100 corms per treatment: Lady Jane	Snow Princess	Rosa Van Lima No. 3	Rosa Van Lima No. 4	Spic & Span	Marg't Fulton
Emmi, 15 minutes	None	94	94	87	87	96	76
Emmi, 2 hours	None	94	90	87	82	89	71
Arasan	None	97	82	90	95	94	79
Corrosive sublimate	None	90	95	94	100	88	64
Aldrin	None	89	99	98	98	85	95
Lindane	None	97	96	98	100	95	94
Heptachlor	None	99	97	95	98	91	95
Emmi + Aldrin	None	96	98	81	64	85	91
Emmi + Lindane	None	79	72	13	9	62	49
Emmi + Heptachlor	None	92	96	98	97	88	87
Arasan + Aldrin	None	96	96	95	100	90	80
Arasan + Lindane	None	94	91	99	96	92	72
Arasan + Heptachlor	None	96	98	97	97	93	82
Emmi, 2 hours	Aldrin	96	97	87	99	95	81
Emmi, 2 hours	Lindane	94	95	90	94	91	78
Emmi, 2 hours	Heptachlor	94	96	98	96	93	75
Arasan	Aldrin	95	99	98	99	89	82
Arasan	Lindane	95	100	93	97	87	72
Arasan	Heptachlor	93	96	99	97	95	71
None	None	87	75	87	91	97	44
Significant difference, 5% level		7	9	9	7	7	15
Significant difference, 1% level		10	12	12	10	10	20

Effects on Flower Production -- Flower yields are shown in Table 2. Effects of the treatments on flower production followed somewhat the same pattern as that shown in the emergence counts, although differences in flower yields were not generally as great as differences in emergence counts. The effects of the Emmi-lindane treatment were still apparent, the flower yield being reduced to zero in one of the Rosa Van Lima plots.

Effects on Corm Production -- Greater production of new corms as a result of treatments was apparent in only two of the plots. In Snow Princess all treatments except the 15-minute soak in Emmi and the Arasan treatment resulted in significantly higher corm yields than those obtained from untreated corms. In the Margaret Fulton plot the Emmi-heptachlor soak and the Emmi soak followed by lindane soil treatment resulted in significantly better yields than those obtained when no treatment was used. The Emmi-lindane corm treatment resulted in reduced yields in three of the plots; the Emmi-aldrin corm treatment resulted in reduced yields in one plot.

Effects on Scab Development -- In order to evaluate fairly the comparative effectiveness of the treatments, corms were graded into four classes at cleaning time as follows: class 1, no scab; class 2, light scab -- corms having 1 to 5 scab lesions; class 3, moderate scab -- corms having 6-10 scab lesions; class 4, severe scab -- corms having more than 10 lesions. From these data a scab severity index for each replicate was calculated by use of a modification of McKinney's (2) formula. In this formula

Table 2. Effects of corm and soil treatments on flower production.

Corm treatment	Soil treatment	Number of flowers produced from 100 corms per treatment: Lady Jane	Snow Princess	Rosa Van Lima No. 3	Rosa Van Lima No. 4	Spic & Span	Marg't Fulton
Emmi, 15 minutes	None	90	94	93	63	102	99
Emmi, 2 hours	None	86	90	83	54	106	94
Arasan	None	94	82	93	67	103	93
Corrosive sublimate	None	90	95	98	64	95	78
Aldrin	None	90	99	101	77	104	102
Lindane	None	101	96	106	79	104	102
Heptachlor	None	97	97	101	80	98	94
Emmi + Aldrin	None	96	98	79	50	104	107
Emmi + Lindane	None	79	72	3	0	71	83
Emmi + Heptachlor	None	91	96	107	81	112	116
Arasan + Aldrin	None	100	96	107	81	117	100
Arasan + Lindane	None	88	91	104	73	93	89
Arasan + Heptachlor	None	105	98	101	79	107	101
Emmi, 2 hours	Aldrin	95	97	103	69	111	97
Emmi, 2 hours	Lindane	94	95	93	60	103	105
Emmi, 2 hours	Heptachlor	96	96	103	77	112	98
Arasan	Aldrin	94	99	100	82	108	92
Arasan	Lindane	90	100	98	64	104	100
Arasan	Heptachlor	93	96	108	77	114	104
None	None	87	75	85	61	103	78
Significant difference, 5% level		11	9	14	17	13	20
Significant difference, 1% level		15	12	19	23	18	27

$$\text{Scab severity index} = \frac{N_1 0 + N_2 1 + N_3 2 + N_4 3}{3t} \times 100$$

where N_1, N_2, N_3, N_4 = number of corms in disease classes 1, 2, 3, and 4 respectively.

t = total number of corms.

Average scab severity on new corms from all plots is shown in Table 4. Greatest benefits from treatments were obtained in the Lady Jane and Snow Princess plots. Scab development in the non-treated checks was greatest in these two plots. An outstanding point was the relative ineffectiveness of the two-hour soaks in corrosive sublimate and Emmi. Heretofore these two treatments have been considered the best available control measures for scab.

The most outstanding feature in the results obtained in this experiment is the reduction in scab development following use of the insecticides as soil treatments in the Lady Jane and Snow Princess plots. As shown in Figures 1 and 2, application of insecticides to the soil was more effective in reducing scab development than was use of the insecticides in the corm treatments. Also, in these two plots the insecticides mixed with Arasan and applied to the corms dry were more effective than the same insecticides used in the emulsion form as wet treatments, either alone or combined with Emmi.

In the Rosa Van Lima No. 3 plot all treatments significantly reduced scab development (Table 4). In the Rosa Van Lima No. 4 plot all corm treatments except Arasan, corrosive sublimate, 15-minute soak in Emmi, and Emmi plus aldrin resulted in less scab than the amount in the non-treated check.

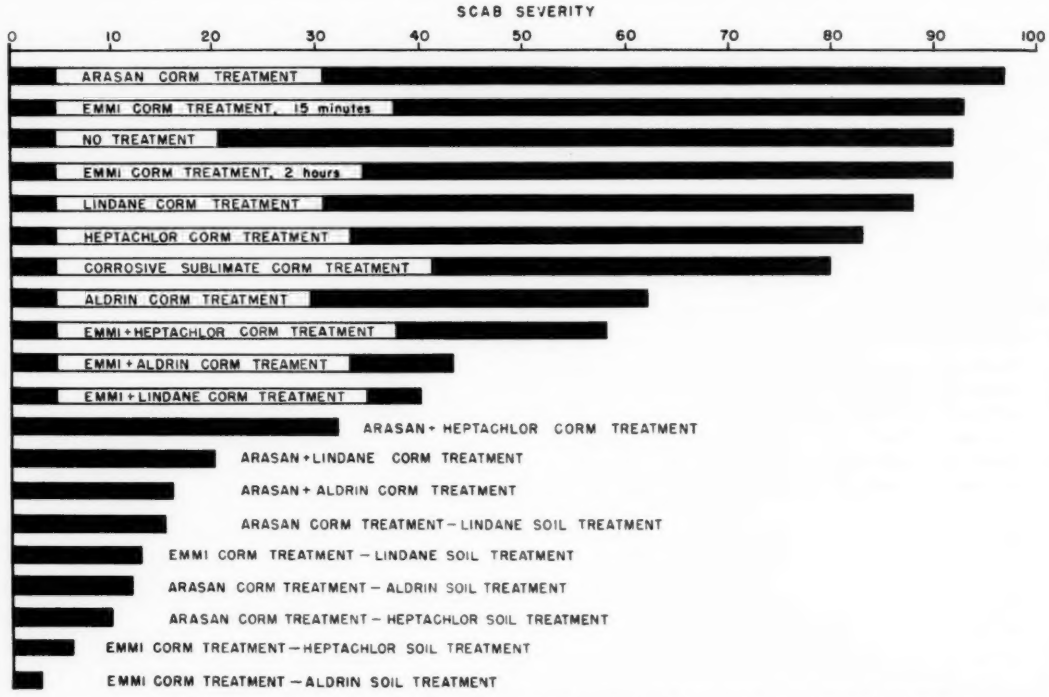

FIGURE 1. Average scab severity on new corms of Lady Jane gladiolus from plot in which corm and soil treatments were used.

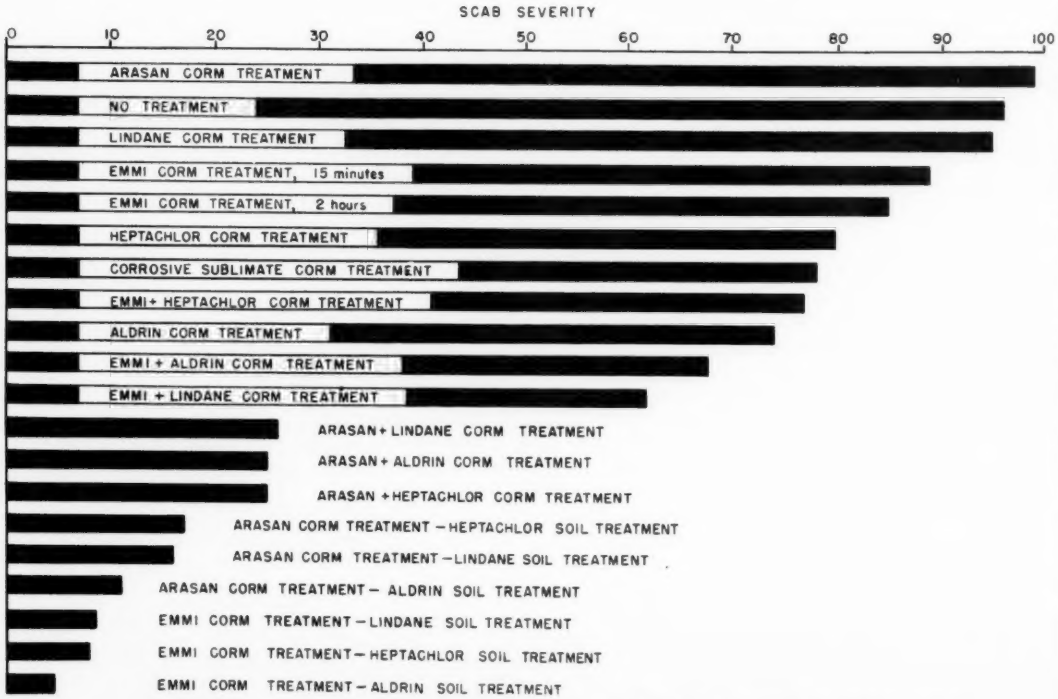

FIGURE 2. Average scab severity on new corms of Snow Princess from plot in which corm and soil treatments were used.

Table 3. Effects of corm and soil treatments on corm production.

Corm treatment	Soil treatment	Number of corms produced from 100 corms per treatment: Lady Jane	Snow Princess	Rosa Van Lima No. 3	Rosa Van Lima No. 4	Spic & Span	Marg't Fulton
Emmi, 15 minutes	None	94	64	107	99	106	105
Emmi, 2 hours	None	95	83	119	97	114	107
Arasan	None	98	51	111	103	106	102
Corrosive sublimate	None	102	93	117	99	105	91
Aldrin	None	100	96	113	100	107	101
Lindane	None	107	87	119	98	104	102
Heptachlor	None	98	88	115	98	101	103
Emmi + Aldrin	None	100	96	103	61	110	110
Emmi + Lindane	None	98	84	20	8	91	97
Emmi + Heptachlor	None	96	83	117	104	115	120
Arasan + Aldrin	None	103	91	115	98	118	99
Arasan + Lindane	None	97	87	114	95	96	95
Arasan + Heptachlor	None	112	96	111	104	112	106
Emmi, 2 hours	Aldrin	98	96	111	101	116	102
Emmi, 2 hours	Lindane	106	98	114	89	111	115
Emmi, 2 hours	Heptachlor	106	102	128	98	116	107
Arasan	Aldrin	97	94	112	101	112	97
Arasan	Lindane	98	94	101	93	103	102
Arasan	Heptachlor	104	96	117	99	120	106
None	None	97	57	108	95	108	93
Significant difference, 5% level		None	16	15	10	13	18
Significant difference, 1% level		None	21	20	13	18	23

In the Spic and Span plot none of the insecticides used alone as corm treatments reduced scab development significantly. Neither did Arasan nor the 15-minute soak in Emmi. All other treatments reduced scab development.

Results in the Margaret Fulton plot were not so good as the results obtained in the other plots (Table 4). Considerable Stromatinia dry rot developed and made scab readings difficult in this variety. No other explanation can be made for the apparently poor results obtained in this plot.

DISCUSSION AND CONCLUSIONS

Results obtained in this experiment clearly demonstrate that bacterial scab can be reduced greatly by use of insecticides. These results help to confirm the supposition of Young (3) that the bacterial scab organism is spread by soil insects. The kind of insects that may be involved is unknown. White grubs are quite common in many of the gladiolus fields in Kankakee County. Soils heavily infested with white grubs nearly always produce scabby corms. Soil samples taken from the gladiolus test plots last summer were carrying large populations of undetermined species of nematodes. It is not inconceivable that nematodes instead of insects might be the agents responsible for spreading scab. Determination of the types of agents involved and how they bring infection about are matters for future investigation.

The dry Arasan-insecticide corm treatments were more effective than the Emmi-insecticide wet treatments, probably because more of the dry insecticide adhered to the corms and thus afforded better protection against attacks of soil insects. It is doubtful if the Arasan had any effect on the scab organism.

Effectiveness of the three insecticides, aldrin, lindane, and heptachlor, used in this experiment did not vary greatly but some difference in their safety was noted. Lindane, when mixed with Emmi and used as a corm treatment, resulted in severe injury to Rosa Van Lima and in

FIGURE 3.

FIGURE 4.

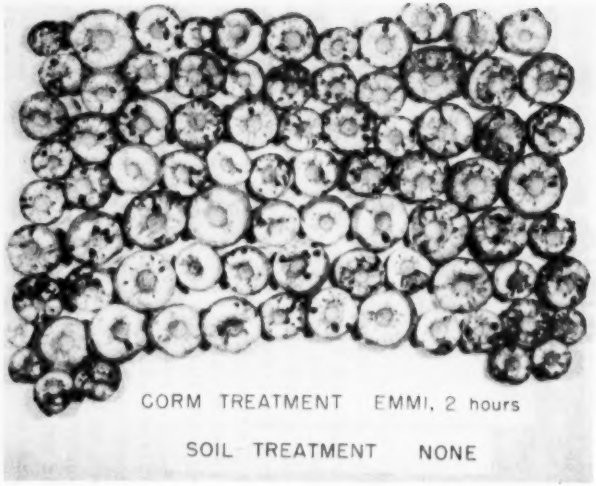

FIGURE 5.

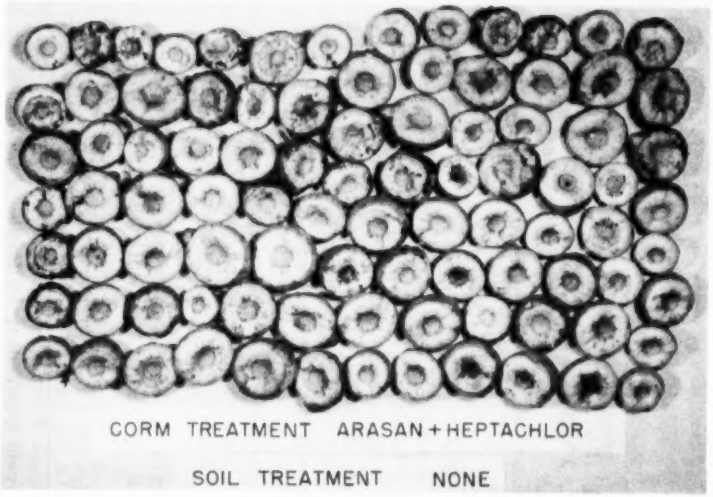

FIGURE 6.

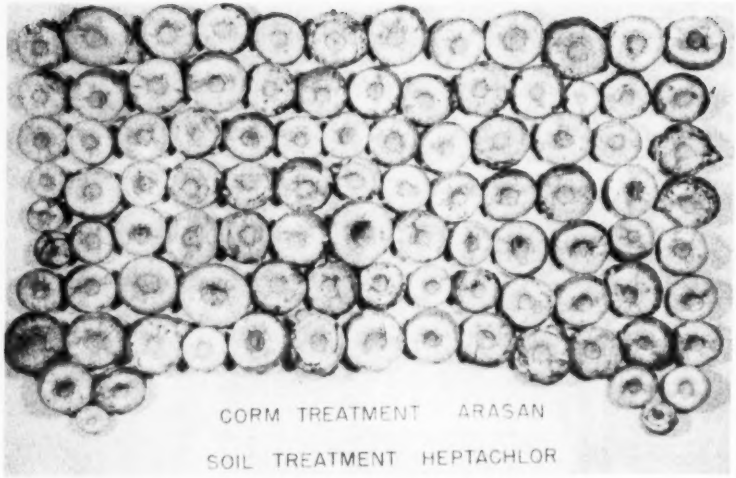

FIGURE 7.

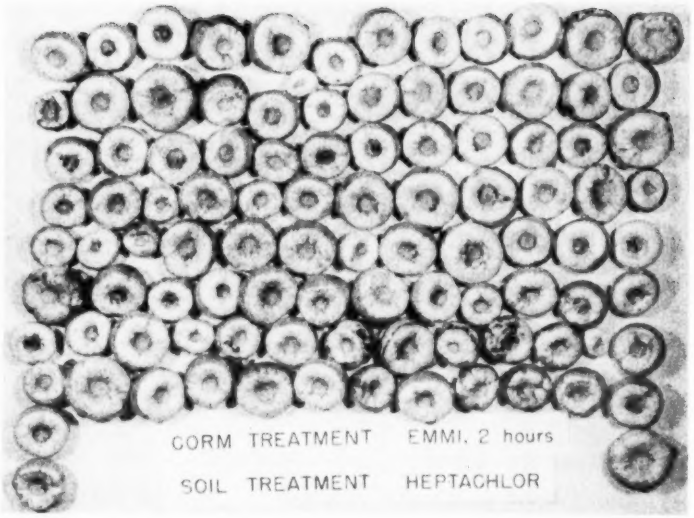

FIGURE 8.

Corms produced from 100 Snow Princess corms given the treatments indicated.

FIGURE 3. A poor yield of scabby corms resulted when no treatment was used. FIGURE 4. This corm treatment resulted in a good yield but most of the corms were scabby. FIGURE 5. This corm treatment resulted in a poor yield of scabby corms. FIGURE 6. Compare yield and amount of scab with that shown in Figure 5. FIGURE 7. A good yield of mostly clean corms resulted from these treatments. FIGURE 8. A good yield of mostly clean corms.

Table 4. Effects of corm and soil treatments on scab development.

Corm treatment	Soil treatment	Average scab severity * on new corms					
		Lady: Jane	Snow Princess	Rosa Van Lima No. 3	Rosa Van Lima No. 4	Spic & Span	Marg't Fulton
Emmi, 15 minutes	None	92.9	89.4	13.4	18.3	37.7	40.8
Emmi, 2 hours	None	92.1	85.4	11.2	10.6	18.5	33.6
Arasan	None	97.2	98.8	13.7	20.1	59.6	53.4
Corrosive sublimate	None	80.3	77.7	12.7	18.3	14.9	30.4
Aldrin	None	62.0	73.6	3.8	6.5	36.6	46.6
Lindane	None	88.5	94.7	5.5	11.0	32.2	43.5
Heptachlor	None	82.6	79.7	9.5	6.5	66.9	60.0
Emmi + Aldrin	None	43.4	68.4	2.5	15.7	13.3	24.8
Emmi + Lindane	None	39.7	62.3	8.3	0.0	7.6	16.3
Emmi + Heptachlor	None	58.4	76.9	6.1	3.9	24.4	36.5
Arasan + Aldrin	None	15.8	24.7	2.5	9.1	24.1	29.0
Arasan + Lindane	None	20.5	26.4	0.3	5.2	7.0	17.3
Arasan + Heptachlor	None	32.4	24.7	0.0	1.7	18.9	23.0
Emmi, 2 hours	Aldrin	2.7	4.5	1.1	0.0	5.5	20.9
Emmi, 2 hours	Lindane	12.8	8.5	0.3	0.3	9.7	13.2
Emmi, 2 hours	Heptachlor	6.1	7.8	1.5	2.9	11.9	19.7
Arasan	Aldrin	12.1	11.1	0.5	0.6	21.9	25.2
Arasan	Lindane	14.7	15.7	2.5	6.7	10.6	22.2
Arasan	Heptachlor	10.3	16.8	2.0	7.6	16.0	13.3
None	None	92.2	95.8	17.8	24.6	43.8	35.0
Significant difference, 5% level		10.4	12.7	2.7	11.2	13.3	14.8
Significant difference, 1% level		14.1	17.1	3.6	14.9	17.7	19.8

*Scab severity was determined by use of a formula as explained in the text.

some injury to Spic and Span. The injury was evidenced by the smaller number of plants emerging and by the poorer yields of flowers and corms following use of this treatment. The Emmi-aldrin corm treatment also appeared to cause similar, though less severe, injury. No evidences of injury were apparent following use of Emmi, lindane, or aldrin alone. Thus it appears that Emmi is not compatible with lindane or aldrin if the mixture is used as a corm dip. When corms were treated with Emmi and the soil was treated with aldrin or lindane no injury resulted.

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SECTION OF APPLIED BOTANY AND PLANT PATHOLOGY, ILLINOIS STATE NATURAL HISTORY SURVEY, URBANA, ILLINOIS

RESPONSE OF AGED VEGETABLE SEED TO SEED TREATMENT[1]

V. R. Wallen, M. Angela Wallace, and W. Bell[2]

Work done in 1952 and 1953 by Wallen and Skolko (1, 2) demonstrated that certain seed treatments brought about a significant increase in the emergence of seedlings of several kinds of vegetables from seed that had been stored for a number of years.

In 1954 experiments were repeated with seeds of the same kinds and varieties of vegetables, except that a different variety of peas was used and watermelon was added. Seed from the same supply was used wherever possible, but it was necessary to make some substitutions where the original samples of aged seed were depleted. Five of the fungicides included in the previous tests were used again and Orthocide 75 and B856C were added. With the small-seeded vegetables 2-ounce samples were subjected to each dosage of fungicide. Larger samples were used with the larger-seeded kinds. The seeds to be treated were placed in wide-mouthed 1000 ml. Erlenmeyer flasks which had been conditioned with the fungicide under test. Known amounts of the fungicide were then placed in the flasks according to the dosage and the flasks were shaken on a mechanical shaker for 5 minutes. The treated seed was stored at room temperature in stoppered flasks at 65° to 75° F. for 2 to 3 weeks before being sown in the field. The seed was sown in rod rows at the rate of 100 seeds per row. Each treatment was replicated four times and randomized. Emergence counts in most instances were taken after 2 weeks.

Table 1. List of vegetable seeds used in seed treatment tests.

Kind	Variety	Source of seed	Year of Harvest
Asparagus	Eden	Ottawa, Ont.	1953
Soybean	Blackeye	Ottawa, Ont.	1948
Beet	Detroit Dark Red #16	Univ. of Man., Man.	1944
Bush Bean	Bountiful	Saanichton, B. C.	1948
Cabbage	Penn. State Ballhead	Ste. Clothilde, Que.	1951
Carrot	Amsterdam	Ste. Clothilde, Que.	1950
Citron	Green Seeded	Summerland, B. C.	1947
Corn	Banting	Ste. Clothilde, Que.	1950
Cucumber	Delcrow	Ottawa, Ont.	1948
Dill	Mammoth	Vancouver, B. C.	1950
Parsley	Champion Moss Curled	Vancouver, B. C.	1948
Muskemelon	Hales Best	Summerland, B. C.	1946
Onion	Brigham Yellow Globe	Ste. Clothilde, Que.	1951
Parsnip	Short Thick	Morden, Man.	1952
Pea	Engress	Ottawa, Ont.	1948
Pumpkin	Sugar	Summerland, B. C.	1948
Radish	French Breakfast	Saanichton, B. C.	1947
Spinach	Bloomsdale L. S.	Saanichton, B. C.	1948
Squash	Kitchenette	Ottawa, Ont.	1951
Swiss Chard	Lucullus	Ottawa, Ont.	1948
Tomato	Bonny Best	Vineland, Ont.	1951
Vegetable Marrow	Long Green Trailing	Agassiz, B. C.	1950
Watermelon	Early Canada	Morden, Man.	1948

The dosages of each fungicide for each kind of vegetable seed are given below:

Arasan (50% tetramethyl thiuramdisulfide): 2 oz. per 100 lb. for pea, soybean, bush bean, citron, corn, cucumber, muskmelon, pumpkin, squash, vegetable marrow, and watermelon; 4 oz. per 100 lb. for asparagus, cabbage, carrot, dill, onion, parsnip, radish,

[1] Contribution No. 1432 from the Botany and Plant Pathology Division, Science Service, Canada Department of Agriculture, Ottawa, Ontario.

[2] Associate Plant Pathologist, Assistant Technician, and Technician, respectively.

Table 2. Mean percentage field emergence from vegetable seed treated with various seed dressings, based on four samples of 100 seeds each.

Kind of seed	Untreated	Arasan	Merculine	Phygon	Vancide 51	Orthocide 75	B856C	Panogen	L.S.D.[a]	
Asparagus	83.25	79.0	78.25	46.5	77.0	75.75	84.0	86.0	6.14	--x[b]
Soybean	19.00	56.00	70.25	58.75	76.25	79.50	59.75	70.50	6.97	
Bush Bean	56.25	50.5	73.75[c]	72.75	71.5	76.25	79.5	78.0	8.14	
Beet	34.0	61.5	53.0	66.0	67.25	69.0	41.0	70.5	11.32	
Cabbage	19.75	27.5	28.0	22.0	19.25	20.75	21.5	24.0	8.74	--x[b]
Carrot	36.5	39.0	34.0	41.75	34.5	40.5	37.25	42.5	5.49	
Citron	4.5	31.5	17.5	45.0	22.0	44.5	18.25	28.0	5.31	
Corn	45.25	63.5	63.0	67.5	69.75	78.75	55.0	64.0	5.32	
Cucumber	0	3.25	4	12.25	4.25	20.0	2.25	11.0	1.94	
Dill	40.75	54.0	39.5	49.5	49.75	41.0	52.5	49.5	7.71	
Parsley	30.0	21.75	25.5	29.0	20.25	27.5	36.75	24.75	6.4	
Muskmelon	7.75	10.0	20.5	18.25	7.25	23.5	13.25	16.5	6.28	
Onion	29.75	42.0	29.5	35.5	39.25	31.0	40.25	32.5	7.94	
Parsnip	25.75	24.0	18.0	22.5	21.5	17.75	26.0	24.75	2.98	--x[b]
Pumpkin	15.75	34.0	77.75	76.25	63.0	80.25	40.25	70.5	11.67	
Pea	10.5	29.25	48.0	44.5	38.5	55.5	42.25	20.5	5.09	
Radish	53.0	71.0	67.25	69.25	68.0	74.25	68.50	60.25	7.16	
Spinach	8.5	39.75	37.0	38.75	38.5	39.75	30.0	35.5	4.95	
Squash	5.5	52	61.25	71.25	9.25	65.75	21.0	52.0	9.99	
Swiss Chard	69.75	84.0	98.0	115.75	115.5	125.75	105.5	117.25	13.77	
Tomato	35.75	51.5	40.25	63.00	27.25	--[d]	54.25	64.5	9.73	
Vegetable Marrow	7.00	69.25	72.0	74.25	29.5	74.0	35.75	56.25	8.22	
Watermelon	2.5	19.25	26.00	57.75	39.25	51.5	10.5	33.25	6.29	

[a] L.S.D. at the 5% level. [b] No significant increase between treatments. [c] Underlined numbers indicate significant increase in emergence. [d] -- Treatment not recommended:

spinach, and tomato; 6 oz. per 100 lb. for parsley; 8 oz. per 100 lb. for beet and swiss chard.

Phygon (98% 2, 3-dichloro-1, 4-naphthoquinone): 1 1/2 oz. per 100 lb. for soybean and corn; 2 oz. per 100 lb. for peas and beans; 3 oz. per 100 lb. for muskmelon; 4 oz. per 100 lb. for asparagus, cabbage, citron, cucumber, onion, pumpkin, radish, spinach, squash, swiss chard, tomato, vegetable marrow, and watermelon; 6 oz. per 100 lb. for parsley and parsnip; 8 oz. per 100 lb. for beet, carrot, and dill.

Panogen (methyl mercury dicyan diamide, 1.4% mercury equivalent): 4 fluid ounces per 100 lb. of seed for all vegetables.

Merculine (emulgate of 10% phenyl mercury salicylate): 2 fluid ounces per 100 lb. seed for all vegetables.

Vancide 51 (sodium salt of dimethyl dithiocarbamic acid + sodium salt of 2-mercapto-benzothiazole): 8 fl. oz. per 100 lb. of seed for pea, soybean, bush bean, and corn; 12 fl. oz. per 100 lb. of seed for citron, cucumber, muskmelon, pumpkin, squash, watermelon, and vegetable marrow; 16 fl. oz. per 100 lb. of seed for asparagus, cabbage, carrot, onion, radish, parsnip, and spinach; 18 fl. oz. per 100 lb. for dill; 24 fl. oz. per 100 lb. of seed for parsley; 32 fl. oz. per 100 lb. of seed for beet and swiss chard; 10% solution dip for tomato.

B856C (1, 3-dichloro-5, 5-diphenylhydantoin): 2 oz. per 100 lb. of seed for bush bean, pea, corn, and soybean; 4 oz. per 100 lb. of seed for asparagus, beet, cabbage, carrot, citron, cucumber, dill, parsley, muskmelon, onion, parsnip, pumpkin, radish, spinach, squash, swiss chard, vegetable marrow, watermelon and tomato.

Orthocide 75 (N-trichloromethylthiotetrahydrophthalimide): 1 1/2 oz. per 100 lb. of seed for corn and cabbage; 2 1/2 oz. per 100 lb. of seed for soybean, pea, and bean; 2 oz. per 100 lb. of seed for watermelon, vegetable marrow, squash, pumpkin, and citron; 3 oz. per 100 lb. of seed for muskmelon, and cucumber ; 6 oz. per 100 lb. of seed for spinach, radish, parsnip, onion, parsley, dill, carrot, and asparagus; 12 oz. per 100 lbs. of seed for swiss chard and beet; no treatment for tomato seed.

Comparison of the results in Table 2 with those obtained in tests of the two previous years (1, 2) reveals that the emergence of seedlings from untreated seed has been drastically reduced in the present test. Beet seed from the same lot was used throughout the three tests. The emergence in the first test (1) was 42.5 percent from the untreated seed, 36.25 percent in the second year (2), and finally 34.0 percent in 1954. Treatment of this seed with all fungicides under test this year resulted in significant increases in emergence. The emergence of untreated corn seed steadily decreased over the 3-year period from a high of 80.8 percent the first year under test to 69.5 percent in the second year to 45.25 percent this year. All treatments this year significantly increased the emergence of this seed. Emergence of soybean harvested in 1948 was 3.25 percent in last year's test and 19.00 percent this year; once again all of the treatments under test this year significantly increased the emergence. With the exception of Arasan all fungicides significantly raised the emergence of bean seed this year. Some kinds of seeds when untreated showed very weak emergence; in particular are pea, cucumber, muskmelon, squash, spinach, vegetable marrow, watermelon, and pumpkin. Most of these seeds, as Table 2 records, responded very well to seed treatment. No significant increase occurred after parsnip, asparagus, and cabbage seed were treated. This finding is in agreement with the results of last year's trial.

Of 23 vegetable seed stocks 20 responded to seed treatment. Eighteen vegetables gave a significant increase in emergence as a result of treatment with Panogen and B856C, 17 with Phygon, 16 with Arasan, and 15 with Merculine, Orthocide 75, and Vancide 51.

In general, if aged seed must be used for the growing of vegetables, in most instances seed treatment will greatly increase the emergence of the seedlings.

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BOTANY AND PLANT PATHOLOGY LABORATORY, SCIENCE SERVICE, CANADA DEPARTMENT OF AGRICULTURE, OTTAWA, ONTARIO

SOME PHYTOTOXIC EFFECTS OF FIVE ACTINOMYCETE ANTIBIOTICS

D. Pramer[1] and Joyce M. Wright

Though a number of reports (References 1 to 13) describe effective disease control resulting from the treatment of seeds with antibiotics, little information is available concerning the influence of antibiotics on seed germination and seedling development. Wright (14) investigated the phytotoxic effect of 12 fungal antibiotics and the actinomycete antibiotic, streptomycin. Alternaric acid, glutinosin, mycophenolic acid, and gliotoxin were the more toxic of the antibiotics studied. The least toxic were griseofulvin, pencillin, and streptomycin. The present communication describes some phytotoxic effects of five actinomycete antibiotics.

MATERIALS AND METHODS

The antibiotics tested were chloramphenicol (chloromycetin), chlortetracycline (aureomycin), neomycin, oxytetracycline (Terramycin) and streptomycin. All except neomycin were crystalline preparations. Antibiotic concentrations of 500, 100, and 0 p.p.m., were prepared in nutrient solution of pH 4 to 5. Seeds of white mustard, red clover, wheat, and cucumber were sown across the diameter of Petri dishes containing filter paper moistened with 10 ml. of the solution to be tested. Prepared plates were stacked vertically and incubated at room temperature in the light. Each plate contained 10 to 12 wheat or cucumber seeds or 15 clover or mustard seeds. All treatments were replicated. After 6 days, the number of seeds that had germinated was recorded, and the amount of root growth was measured. The measure of root growth was fresh weight in the case of wheat and cucumber and root length in the case of mustard and clover.

RESULTS AND DISCUSSIONS

The results are presented in Table 1. The percentage germination of the seeds was not significantly influenced by any treatment, but in all cases inhibition of root (and to a lesser extent of shoot) growth occurred. No stimulation of root growth or formative effects resulting from antibiotic treatment were observed.

Table 1. Toxicity of various antibiotics.

Antibiotic	Root growth as percent of control			
	Mustard	Red clover	Cucumber	Wheat
500 p.p.m.				
Neomycin	40	34	22	57
Streptomycin	39	42	21	53
Oxytetracycline	12	12	14	16
Chlortetracycline	9	4	14	22
Chloramphenicol	13	18	9	16
100 p.p.m.				
Neomycin	87	74	72	63
Streptomycin	80	61	54	46
Oxytetracycline	34	39	19	38
Chlortetracycline	30	38	23	37
Chloramphenicol	30	37	17	24

[1]Present address: Department of Agricultural Microbiology, New Jersey Agricultural Experiment Station, Rutgers University, New Brunswick, New Jersey.

The greater toxicity of chloramphenicol, oxytetracycline, and chlortetracycline compared with streptomycin and neomycin was indicated by a more pronounced inhibition of root development and chlorophyll formation: no chlorophyll was produced in mustard seedlings whilst other seedlings either reacted in a similar manner or, at the best, were pale green or variegated. Wheat was least affected by the antibiotics, none of which completely suppressed chlorophyll formation although very little was produced in the presence of chloramphenicol. On the other hand, mustard was very sensitive, chlorophyll formation being completely inhibited by chloramphenicol, chlortetracycline, and oxytetracycline, and by streptomycin at the higher concentration. Anthocyanin, which is present in the young cotyledons of mustard seedlings, was not formed in those seedlings treated with chloramphenicol. Neomycin had no effect on the formation of chlorophyll in any of the seedlings at the concentrations tested.

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IMPERIAL CHEMICAL INDUSTRIES LTD., BUTTERWICK RESEARCH LABORATORIES, WELWYN, HERTS., ENGLAND

ANTIBIOTIC TREATMENT OF SEED POTATOES IN RELATION TO SEED-PIECE DECAY, BLACKLEG, PLANT GROWTH, AND YIELD RATE

Reiner Bonde[1]

Laboratory and greenhouse studies conducted in Maine (1-4) have shown that treating potato seed pieces in solutions of streptomycin sulfate, with or without the presence of Terramycin hydrochloride, was effective in reducing both blackleg and the seed-piece decay caused by blackleg bacteria (Erwinia atroseptica).

Experiments were conducted during 1954 to determine if antibiotic treatments also would control seed-piece decay and blackleg under field conditions. In addition, information was obtained on the effect of the antibiotic treatments on some phases of plant growth and on yield rate.

EXPERIMENTS WITH AGRIMYCIN

Experiments were conducted in cooperation with potato growers to test the value of "Agrimycin" seed treatments[2] under farm conditions.

The farmers used their healthy commercial seed stocks for these tests. The seed potato tubers were cut in the usual manner and the seed pieces were treated immediately in Agrimycin solutions at a streptomycin concentration of 100 ppm (9.34 oz. Agrimycin per 100 gal. water).

One lot of each farmer's seed stock was given an "instant dip" treatment, another similar lot was immersed or "soaked" 30 minutes, and a third was cut but not treated and was used as the control. The three cut seed lots were stored under farm conditions for periods varying from 1 to 3 weeks before being planted in the field.

The seed lots were planted by machine in the usual manner. Observations were made on the condition of the seed lots before being planted, on the emergence in the field, and on the development of blackleg. Yield rates were determined at the time of harvest.

Control of Seed-Piece Decay. All of the treated seed stocks remained firm and had a bright color and no seed-piece decay. In contrast, from 5 to 10 percent of the untreated seed pieces on the different farms had a shallow surface spotting caused by bacteria.

Effect on Emergence, Blackleg, and Yield Rate. Table 1 summarizes the percentage of plants with blackleg, and the yield rates.

In the Fort Kent seed lot the 30-minute treatment resulted in a marked reduction in the emergence percentage[3]. Also, the Fort Kent field was very wet and, further, became flooded by heavy hurricane rain, which resulted in no crop being produced. It was noted, however, that the plants from the seed pieces given the instant dip treatment withstood the wet soil conditions longer before dying than the plants from the untreated seed pieces. It was apparent that this treatment protected the plants somewhat from the injury caused by the wet soil conditions.

The data in Table 1 also show that both the instant and the 30-minute treatments increased the number of plants that emerged in the fields located at Houlton, Patten, and Presque Isle. The parts of the fields planted with the treated seed were superior in all three locations, the plants being greener, more uniform in size and more vigorous. Figure 1 shows the effect of treating Katahdin seed pieces on stand and vigor. The treatments also greatly reduced the percentage of blackleg plants in each field. Blackleg was absent from the treated-seed areas earlier in the season but began to appear in trace amounts toward the end of the growing period. One farmer noted that treatment increased the number of eyes that produced sprouts.

All of the Agrimycin treatments increased the yield rate. This increase varied from 19 bushels (7 barrels) per acre for the instant dip treatment in the Patten field to 72 bushels per acre (26 barrels) for the 30-minute treatment in Presque Isle.

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[1] Plant Pathologist, Maine Agricultural Experiment Station, Orono, Maine.

[2] Agrimycin contains 15% streptomycin and 1.5% Terramycin and is manufactured by Chas. Pfizer & Co., Inc., Brooklyn, New York.

[3] The writer has not observed this effect from treating freshly cut seed potatoes in Agrimycin solutions elsewhere.

Table 1. Effects at various locations in Maine of treating farmer's seed potatoes in Agrimycin solution.

Location	Treatment[a]	Emergence %	Blackleg %	Yield per acre[b] Bbls.	Yield per acre[b] Bus.	Increase in yield rate Bbls.	Increase in yield rate Bus.
Fort Kent	Instant dip[c]	95	--	--	--	--	--
	30-minute soak	20	--	--	--	--	--
	No treatment	85	--	--	--	--	--
Houlton	Instant dip[c]	94	Trace	200	550	11	30
	30-minute soak	95	Trace	200	550	11	30
	No treatment	89	2.0	189	520	--	--
Patten	Instant dip[c]	96	Trace	161	443	7	19
	30-minute soak	95	Trace	162	446	8	22
	No treatment	90	6.0	154	424	--	--
Presque Isle	Instant dip[c]	95	Trace	240	660	25	69
	30-minute soak	97	Trace	241	663	26	72
	No treatment	83	7.2	215	591	--	--

No statistical comparisons made

[a] Freshly cut farmers' seed potatoes were treated in solution with streptomycin concentration of 100 ppm and placed immediately in barrels in farm storage for periods of from 1 to 3 weeks before planting in the field.

[b] Yields were not obtained for the Fort Kent experiment because the field was flooded by hurricane-brought water which destroyed the plants.

[c] Immersed in solution long enough to become completely moist on all surfaces.

Table 2. Effects of treating freshly cut potato seed pieces in streptomycin nitrate solutions; Maine experiments, 1954.

Comparisons made[a]	Control	50 ppm	100 ppm
Height of plants (cm.) Aug. 26	42.5	44.2	38.9[b]
Number of stems per plant	3.8	4.7[c]	4.4[d]
Number of flower clusters per plant	4.7	5.6	5.8
Weight of foliage of 25 plants Sept. 20 (lbs.)	12.1	16.9[e]	15.3
Yield per acre -- Barrels	105.5	139.6[e]	128.5
Bushels	290.1	383.9[e]	353.4

[a] Based on four 25-foot plots per treatment.

[b] Significantly lower than control at 5% level.

[c] Significantly higher than controls at 1% level.

[d] Bordering on significance at 1% level.

[e] Significantly higher than controls at 5% level.

FIGURE 1. Effect on stand and vigor of plants resulting from treating Katahdin seed pieces in streptomycin sulfate 100 ppm solution. Left -- Plants from treated seed pieces that had been inoculated by spraying with blackleg bacteria. Center -- Plants from untreated, uninoculated seed pieces. Right -- Poor stand resulting from planting inoculated seed pieces that had not been treated.

EXPERIMENTS WITH STREPTOMYCIN NITRATE

Another study was conducted on Aroostook Farm, in the Presque Isle area. Here freshly cut seed pieces were treated for 30 minutes in 50- and 100-ppm solutions of streptomycin nitrate. The treated seed pieces and the untreated controls were stored for seven days before being planted.

No decay occurred on the untreated control or treated seed pieces and there was 100 percent emergence. The absence of missing hills afforded an opportunity to observe the effect of streptomycin nitrate on plant growth. Data were obtained on average height, number of stems and flower clusters per plant, total weight of foliage per 25 plants at harvest time, and yield rate. The data are in Table 2.

Increase in Height, Number of Stems, and Flower Clusters per Plant. Observations made during the growing season indicated that the plants from the treated seed pieces were more vigorous than adjacent control plants. The data in Table 2 show that the 50-ppm treatment increased plant height (measured August 26) slightly with respect to the height in the controls and the 100-ppm plots. The 100-ppm treatment retarded the growth of the plants up to August 26. This effect wore off gradually as the season progressed.

The data also show that the 50-ppm treatment significantly increased the number of stems per hill. The 100-ppm treatment increased the number of stems to a less extent. The plants from treated seed pieces had more flower clusters per hill than the controls.

Increase in Weight of Foliage and in Yield Rate. The data in Table 2 show that both the 50- and the 100-ppm treatments increased the weight of the plant foliage, the former signficantly.

There was a significant increase in the yield rate in the 50-ppm plots, and a rather substantial increase in the 100-ppm plots.

The increase in plant vigor and in yield rate suggests that there was a physiological stimulation by streptomycin nitrate, inasmuch as seed-piece decay was absent at planting time and there were no missing hills. Although the 100-ppm treatment was of considerable benefit when the controls are considered, it was less beneficial than the 50-ppm treatment, for reasons as yet unknown.

SUMMARY AND RECOMMENDATIONS

Treating seed pieces in Agrimycin solutions (streptomycin concentration 100 ppm) on commercial farms reduced the amount of seed-piece decay, generally increased the emergence (stand) in the field, greatly reduced the percentage of blackleg, generally improved the color, size and vigor of the plants, and always increased the yield rate. The instant-dip treatment as used on the farms was as effective as the 30-minute soak treatment in reducing seed-piece decay and blackleg. On one farm the 30-minute soak treatment reduced the emergence percentage considerably.

Treating cut seed potatoes in streptomycin nitrate solutions at concentrations of 50 and 100 ppm increased the plant height, the number of stems per hill, the number of flower clusters per plant, the weight of the foliage, and the yield rate. Streptomycin nitrate at 100 ppm was somewhat less beneficial than at 50 ppm.

Although the results reported in this paper indicate that treating cut seed potatoes in streptomycin solutions may be beneficial, they are not conclusive and studies should be conducted on a larger scale before recommending this treatment for general farm use.

It is recommended that farmers who are interested in testing antibiotics for the control of seed-piece decay and blackleg should test the Agrimycin 100-ppm instant dip treatment on a rather small scale.

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MAINE AGRICULTURAL EXPERIMENT STATION, ORONO

CONTROL OF STEM RUST OF WHEAT WITH ANTIBIOTICS[1]
I. GREENHOUSE AND FIELD TESTS

V. R. Wallen[2]

Abstract

Several antibiotics, actidione, trichothecin, gliotoxin, helixin B, endomycin, and candicidin, were used as sprays in the greenhouse for the control of stem rust of wheat. Actidione, the most promising in greenhouse trials, was used in the field at concentrations of 25, 50, 100, 200, and 500 p.p.m. Although applications at concentrations of from 50 to 500 p.p.m. produced symptoms of phytotoxicity in the form of leaf injury, the plants appeared to recover in most cases. The degree of infection varied from 100 percent in the control to 0 to 5 percent in the plot sprayed every 10 days at 500 p.p.m. Plots sprayed every 10 days, sprayed before heading, and sprayed after heading consistently outyielded unsprayed control plots.

The control of rust diseases of cereal crops by means of applications of chemicals to the soil or plants has been attempted by several investigators. Greaney (3) obtained effective control of stem rust of wheat and oats by dusting the plants with a finely divided sulfur at the rate of 30 lb. per acre at each application, the number of applications and the amount of sulfur varying with the severity of rust infection. Gigante (2) recommended borax for the control of leaf rust (Puccinia triticina) and stripe rust (P. glumarum). Gassner and Hassebrauk (1) and Hassebrauk (5) tested a large number of chemicals for the control of cereal rusts by applying the chemicals to the soil at the time of rust infection. They suggested that the most promising chemicals were acridin, 1 bromo-2 naphthol, picric acid, and alpha-naphthol. Hart and Allison (4) found that toluenesulfonylamide compounds were effective in greenhouse tests. The infection of wheat stem rust was reduced from 90 percent in the check series to 20 percent with the toluene compounds; however, large quantities of these compounds were necessary to attain this degree of control. Hassebrauk (6) used various chemicals against four rusts: P. triticina, P. simplex, P. coronata, and P. glumarum. These chemicals were applied to the soil when the plants were inoculated. Several chemicals, para-amino benzol sulfonamide, acetyl sulfonilamide, and saccharin gave good control. Hotson (7) in greenhouse tests found that sulfadiazine gave excellent control of stem rust of wheat applied at the rate of 4 mg. per pot in aqueous solution applied to the soil. Continuing this work Hotson (8) found that certain sulfa drugs will control wheat stem rust when they are applied as a post-infection spray at the rate of 5 lb. per acre. Silverman (10) found that Acti-dione at 20-25 parts per million controlled stem rust of wheat by reducing the number of pustules and by delaying sporulation.

I. Greenhouse Tests

In preliminary greenhouse tests wheat seedlings of the variety Little Club were thinned out to ten seedlings per pot. After the plants were approximately 4 inches high they were inoculated with race 15B of Puccinia graminis var. tritici. Inoculum was maintained on older plants of Little Club and the inoculation of the seedling plants was accomplished by transferring urediospores to the leaves with the thumb and forefinger. The inculated plants were placed overnight in a constant-temperature high-humidity tank at 17° C. The plants were removed and then placed on benches in the greenhouse. Forty-eight hours later the plants were sprayed with 10 ml. of the following antibiotics: actidione, trichothecin, gliotoxin, helixin B, candicidin, and endomycin. Two pots containing 10 seedlings were used for each treatment. The antibiotics were applied as a spray, 10 ml. per pot, by means of a de Vilbiss #251 atomizer. In a second set of tests the plants were sprayed with the various antibiotics 48 hours before inoculation with the rust spores.

All the plants were examined daily for the appearance of rust pustules and final readings were taken after 25 days.

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1 Contribution No. 1433 from the Botany and Plant Pathology Division, Science Service, Canada Department of Agriculture, Ottawa, Ontario.

2 Associate Plant Pathologist.

Table 1. Effect of various concentrations of antibiotics on stem rust development on Little Club wheat in greenhouse tests.

Treatment	Rust rating									
	Pre-inoculation spray (p.p.m.)					Post inoculation spray (p.p.m.)				
	25	50	100	200	500	25	50	100	200	500
Actidione	-	-	-	-	-	+	+	-	-	-
Gliotoxin	+	+	+	+	+	+	+	+	+	+
Trichothecin	+	+	+	+	-	+	+	+	+	+
Candicidin	+	+	+	+	+	+	+	+	+	+
Endomycin	+	+	+	+	+	+	+	+	+	+
Helixin B	+	+	+	+	+	+	+	+	+	+

+Indicates presence of rust.

- Indicates rust not present.

Table 1 shows that actidione was effective in controlling rust in the pre-inoculation spray schedule at all concentrations used. In the post-inoculation spray, actidione controlled stem rust at concentrations of 100 p.p.m. and greater. Trichothecin was effective as a pre-inoculation spray at 500 parts per million. Some antibiotics caused phytotoxicity in the form of tip and blade injury and dieback of the leaf tissue.

II. Field Test

Because of the excellent results obtained with Acti-dione in the preliminary greenhouse trials, this antibiotic was selected for field trials on the control of stem rust of wheat.

Small plots, each consisting of nine rod rows, were seeded with Garnet spring wheat, 100 seeds to a row. The purpose of the light seeding was to permit easy observation of the plants for rust rating and to make sure that the plants were covered with the antibiotic when they were sprayed. Five plots were sown in early June and 20 other plots were sown later in the season.

The spray schedule for the plants was as follows: 10 days after emergence, every 10 days, just before heading, and just after heading. Concentrations of 25, 50, 100, 200, and 500 parts per million were used. For the experiment 20 plots were sprayed with the antibiotics and five were unsprayed. A 5-gallon pressure sprayer was used to apply the actidione solutions. The Acti-dione was applied at the rate of one liter of solution per nine-row plot.

The plots were all inoculated with a mixture of races of Puccinia graminis var. tritici when the plants were approximately 30 days old. Urediospore inoculum was mixed with Merck talc, 1 part spores to 100 parts talc. The mixture was applied to one of the outside rows of each plot by means of a small hand duster. At maturity the rust had spread evenly throughout the plot. Natural infection of the plants was probably responsible for most of the rust present.

The plants were rated for percentage of rust at maturity, according to the method of Peterson et al. (9), and yields in grams of the various plots were also taken.

From Table 2 it can be seen that increasing the concentration of Acti-dione in repeated applications every 10 days steadily reduced the level of rust infection. A spray applied just before heading at 50 p.p.m. and concentrations above this level also reduced the rust intensity. Sprays applied after heading reduced the rust slightly at 100 and 200 p.p.m., and by 50 percent at the 500 p.p.m. level. Sprays applied 10 days after emergence did not appear to reduce the rust intensity.

Evidence of phytotoxicity was noted at concentrations as low as 50 p.p.m. The symptoms were a general lighter green color of the whole plant and on individual plants a dieback of the tips of the leaves. Despite this evident phytotoxicity the plants appeared to recover and the yields of sprayed plots were as high as, and in some cases higher than, the yields of the control plots.

Table 2. Rust percentage and yield in grams of Garnet wheat sprayed with concentrations of 25, 50, 100, 200, and 500 p.p.m. of actidione, field test.

Treatment	p.p.m.	Amount of rust observed	Yield in grams
Control	0	Moderate to heavy 100%	152.0
Sprayed:			
10 days after emergence	25	Moderate to heavy 100%	153.5
Every 10 days	25	Slight to moderate 50-60%	250.25
Before heading	25	Moderate 80-90%	263.75
After heading	25	Moderate to heavy 90%	325.75
Control	0	Moderate to heavy 100%	112.5
Sprayed:			
10 days after emergence	50	Moderate to heavy 100%	158.25
Every 10 days	50	Slight to moderate 30-35%	347.0
Before heading	50	Moderate 75-80%	219.25
After heading	50	Moderate to heavy 90%	244.5
Control	0	Moderate to heavy 90-100%	x475.5
Sprayed:			
10 days after emergence	100	Moderate to heavy 90-100%	x425.75
Every 10 days	100	Trace to moderate 10-15%	x425.75
Before heading	100	Slight to moderate 20-25%	x531.0
After heading	100	Slight to heavy 70-80%	x445.0
Control	0	Moderate to heavy 100%	266.5
Sprayed:			
10 days after emergence	200	Moderate to heavy 100%	166.5
Every 10 days	200	Trace to slight 15-20%	390.5
Before heading	200	Slight to moderate 60%	411.0
After heading	200	Moderate 75%	275.5
Control	0	Moderate to heavy 100%	127.0
Sprayed:			
10 days after emergence	500	Moderate to heavy 100%	125.75
Every 10 days	500	0 to trace 0-5%	227.0
Before heading	500	0 to slight 10%	251.5
After heading	500	Slight to moderate 40-50%	176.25
			Average yields of all treatments
Control	--	--	226.7
Sprayed:			
10 days after emergence	--	--	205.95
Every 10 days	--	--	351.4
Before heading	--	--	335.3
After heading	--	--	293.4

x Higher yields due to earlier planting than other plots.

The two rows on either side of each plot were discarded and yields were taken on the middle five rows. No statistical analysis was attempted in this preliminary trial; however, the plots which were sprayed every 10 days with all concentrations of Acti-dione clearly outyielded the control plots. The spray applied just before heading reduced the amount of rust in most cases and accounted for an increase in yield over that of the control plot in every case.

The seed harvested from each plot was sown in partially sterilized soil in the greenhouse to determine whether Acti-dione used as a spray would affect the germination of the seed produced by the sprayed plants. Over 90 percent emergence was obtained from this seed; therefore no such phytotoxic effect had occurred.

Conclusions

In this preliminary trial an attempt was made to find out if an antibiotic could be used to control stem rust of wheat. Acti-dione appeared to show promise both in greenhouse and field trials; however, all concentrations were purely experimental, and quantity and concentrations of the spray applied may have been considerably more than was necessary. Yields of sprayed plots were consistently greater than those of unsprayed control plots. Emergence of seed from plants sprayed with Acti-dione was over 90 percent in all treatments.

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BOTANY AND PLANT PATHOLOGY LABORATORY, SCIENCE SERVICE, CANADA DEPARTMENT OF AGRICULTURE, OTTAWA, ONTARIO

STREPTOMYCIN FOR FIRE BLIGHT CONTROL ON APPLE IN NORTH CAROLINA

C. N. Clayton

Summary

Five streptomycin formulations were compared for control of fire blight of apple trees in North Carolina in 1954. Streptomycin sulfate plus Terramycin, soluble streptomycin sulfate, and streptomycin nitrate were effective. In three sprays during bloom streptomycin at 100 ppm was more effective than at 50 ppm. Foliage injury appeared a few days after spraying but disappeared after several weeks.

Several investigators (1, 2, 3, 4) have reported that streptomycin sprays on apple or pear controlled fire blight caused by Erwinia amylovora. Therefore, in 1954 five formulations of streptomycin were tested for this purpose in North Carolina.

The materials used were: Agrimycin-100 (15% streptomycin + 1.5% Terramycin) furnished by Chas. Pfizer and Company, Inc.; Liquid Streptomycin Nitrate (11.8% streptomycin) furnished by Mathieson Chemical Corporation; Streptomycin Formulation STS Soluble (54% streptomycin), Streptomycin Formulation STB Insoluble (10% streptomycin), and Streptomycin Formulation STD Insoluble (10% streptomycin) furnished by Merck and Company, Inc. DuPont Spreader-Sticker was included at 2 oz. per 100 gallons of spray with each material. Three spray applications were made during bloom in each of four orchards.

At the McCullers Branch Experiment Station each of the streptomycin materials (Table 1) at 100 ppm of streptomycin was compared on 5-year-old Gallia Beauty apple trees. Each material was applied to six single-tree replications at 400 to 500 lbs. pressure. The first spray was applied on April 20 at 75 percent full bloom, the second on April 27 at petal-fall, and the third on May 11. No fungicide or insecticide was used on the trees. All trees except those in treatment 7 were inoculated on April 29 with a suspension of fire blight bacteria. The bacterial inoculum was applied at 400 to 500 lbs. pressure from a power sprayer.

No blight had developed from natural infection on any tree by petal-fall. However, on May 10, or 12 days after inoculation, blight symptoms were present on a few spurs and terminal shoots. The blighted blossom clusters and terminal shoots on each tree were counted on May 25 and again on June 2 (Table 1). On June 29, after the blight infections had progressed down the branches, counts were made of the cuts per tree necessary to remove all blighted spurs and terminals. Agrimycin, Streptomycin Nitrate, STS, and STD sprays reduced fire blight infections about 50 percent in comparison with the non-sprayed. The STB formulation failed to reduce infection. No blight developed on the non-inoculated trees.

Powdery mildew (Podosphaera leucotricha) was moderate to very heavy on the trees used in this experiment. Ratings of its severity indicated that the streptomycin treatments had no effect on its development.

In another experiment at McCullers Branch Experiment Station the above formulations of streptomycin (Table 2) at 100 ppm streptomycin were used in different fungicide-insecticide sprays in the pink and petal-fall applications, and alone in full bloom, on four single-tree replicates of each of the following varieties: Golden Delicious, Stayman, Red Delicious, Wrixparent, Close, Anoka, and Lodi. In addition, Agrimycin and Streptomycin Nitrate were tested at 50 ppm streptomycin. Very little blight occurred except a light infection in the Golden Delicious planting. The blighted spurs and terminals per tree were counted on June 2. Blight was not controlled completely by any treatment (Table 2); however, all formulations of streptomycin reduced it considerably in comparison with the check trees. Streptomycin at 50 ppm appeared to be less effective than at 100 ppm. There was no apparent incompatibility between streptomycin and ferbam, wettable sulfur, captan, phenyl mercuric acetate, or parathion.

In Wilkes County, Agrimycin and Streptomycin Nitrate were compared in Mr. Clarence Fletcher's orchard of large Golden Delicious trees where blight had been severe in recent years. Each material, at 100 ppm streptomycin, was applied to adjacent blocks that consisted of 3 rows of 18 to 20 trees each. Spray applications were made on April 12, 15 and 19 when the percentages of open blossoms were 20 to 30, 75 to 90, and 90 to 100, respectively. The remainder of the orchard received no sprays for blight control.

Blight first appeared in the orchard on April 23 from infection initiated during rainy weather on April 10. By April 28 blight was severe. It continued to develop during the relatively cool

Table 1. Effect of streptomycin spray treatment on control of fire blight of Gallia Beauty apple trees. McCullers Branch Experiment Station, North Carolina.

Treatment No.	Material[a]	Average number blighted spurs and terminals per tree		Number cuts to remove blighted spurs and shoots per tree, June 29
		May 25	June 2	
Inoculated[b]				
1.	Agrimycin-100	6.3	12.8	13.8
2.	Streptomycin Nitrate	3.8	10.3	11.2
3.	STS	9.5	13.8	13.7
4.	STB	11.3	25.7	29.8
5.	STD	5.7	10.3	12.2
6.	None	12.5	21.7	20.8
Not inoculated				
7.	None	0	0	0
	L.S.D. at 5% level	5.4	3.5	

[a] All streptomycin formulations were used at rate of 100 ppm streptomycin. DuPont Spreader-Sticker at 2 oz. -100 gal. was included with each material.
[b] Inoculated with fire-blight bacteria April 29.

Table 2. Effect of streptomycin sprays on control of fire blight on Golden Delicious apple trees in three orchards.

Material	Concentration of streptomycin (ppm)	Average number blighted spurs or terminals per tree		
		Lutz orchard	Fletcher orchard	McCullers orchard
Agrimycin	100	0.4	83	0.7
Agrimycin	50			6.0
Streptomycin Nitrate	100		199	0.5
Streptomycin Nitrate	50			8.0
STS	100			2.0
STB	100			6.0
STD	100	0		1.8
Check	0	18.3	941	17.5
L.S.D. (5% level)		--	96	5.9
L.S.D. (1% level)		--	129	8.4

FIGURE 1. Left -- Fire blight on an untreated Golden Delicious apple branch. Right -- branch sprayed 3 times during bloom with 100 ppm streptomycin.

wet May and early June, with many infections occurring in the tips of succulent shoots. The blight epidemic on the non-treated part of the orchard was one of the worst on record; 50 percent or more of the spurs and shoots were killed by blight. (Fig. 1). On May 12 the number of blighted spurs and terminals on 15 check trees averaged 941 per tree, on 15 Agrimycin-treated trees 83, and on 15 Streptomycin Nitrate-treated trees 199. Thus, control of fire blight with Agrimycin averaged 91 percent and with Streptomycin Nitrate 79 percent in comparison with the non-treated check (Table 2).

In Mr. Everett Lutz's orchard, in Cleveland County, Agrimycin and STD at 100 ppm streptomycin were tested on large Golden Delicious trees. Blight had been severe in this orchard in previous years. Forty to 50 trees in adjacent blocks were treated with each material. Spray applications were made on April 9, 12, and 16, when the percentage of open blossoms averaged approximately 20, 60, and 100, respectively. On May 12 the average number of blighted spurs and terminal shoots was 18.3 for the check, 0.4 for Agrimycin, and 0 for STD (Table 2).

In all tests foliage injury appeared on a low percentage of the leaves a few days after trees were sprayed with streptomycin. The chlorotic symptoms were particularly noticeable at the margins, tips, and midribs of leaves. The injury occurred on all varieties. The degree of injury was about the same for Agrimycin, Streptomycin Nitrate, and STS. Less injury occurred with STD than with STS, and with 50 ppm streptomycin than with 100 ppm. STB caused no chlorosis. The injury was greatest where sprays were heavily applied. The affected leaf areas were at first bright yellow, later became pale yellow, and after several weeks recovered their green color. The injury could not be found after mid-season. Affected leaves did not drop as a result of the injury.

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PLANT PATHOLOGY, NORTH CAROLINA STATE COLLEGE, RALEIGH, NORTH CAROLINA

A COMPARISON OF DIFFERENT FUNGICIDES FOR THE CONTROL OF APPLE POWDERY MILDEW IN 1954

Louis Beraha, Roy A. Wilson, and John C. Dunegan[1]

During the past several years, apple powdery mildew caused by Podosphaera leucotricha has been unusually prevalent in certain blocks of Jonathan and Rome Beauty apples in eastern orchards.

The control of this disease poses a serious problem. Sulfur preparations, long the standard mildew remedies, are no longer being used extensively by commercial growers because of their deleterious effect on apple trees. An effective non-phytotoxic material is therefore urgently needed. In 1954, seven organic compounds were compared with wettable sulfur for the control of apple powdery mildew, in a block of 11-year-old Rome Beauty trees at Plant Industry Station, Beltsville, Maryland. Eighty trees showing over-wintering bud infections were arranged in a randomized 10-treatment, 8-replicate design.

The various treatments (Table 1) were applied on April 13 (pre-pink), April 19 (pink), April 30 (calyx), and May 13 (first cover). Parathion (1/3 pound in 100 gallons of water) was used in all the sprays except the pre-pink application. Tween 20 (0.025%) was added to Actidione as a spreader. The check trees were sprayed with water. Both concentrations of Actidione caused serious leaf injury and this treatment was omitted in the fourth (first cover) spray.

Between June 7 and 15, leaves of approximately uniform age (third leaf from the apex) were collected from 50 shoots selected at random from each tree. These leaves were rated in 6 classes on the basis of approximate area of the underside of the leaf covered by the mildew fungus (Fig. 1). A coded infection grade for each treatment was derived from the sum of the class ratings multiplied by the class frequency divided by 8 (the number of replicates in a treatment).

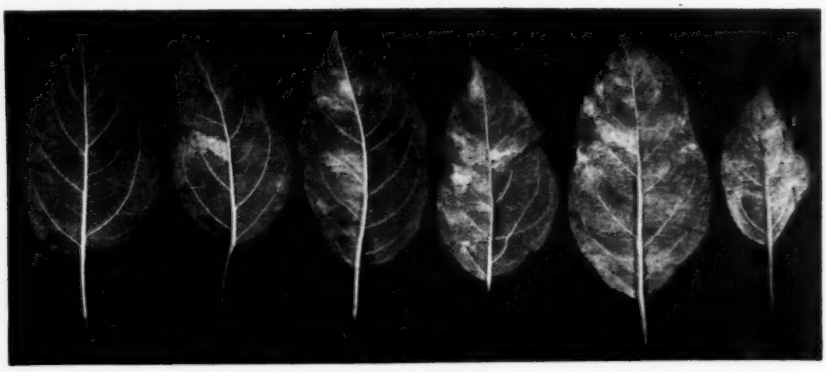

FIGURE 1. Scale used to appraise the approximate leaf area covered by Podosphaera leucotricha.

[1] Respectively, Pathologist, Biological Aid, and Principal Pathologist, Horticultural Crops Research Branch, Agricultural Research Service, U. S. Department of Agriculture, Beltsville, Maryland.

Table 1. Apple mildew on leaves of Rome Beauty trees sprayed with various materials.

Material	Dosage rate	Coded infection grade	Mean percent of lower leaf area infected
Actidione	1 p.p.m.	1920	38.4
Actidione	2 p.p.m.	2463	49.3
Karathane	3/4 lb: 100	936	18.7
Captan	2:100	2873	57.5
Zineb	1:1/2:100	3188	63.8
*Vancide Z65 (zinc salt)	3:100	2349	47.0
*Vancide F995W (manganese salt)	2:100	1604	32.0
*Vancide F956W (iron salt)	2:100	2260	45.2
Wettable sulfur	6:100	399	8.0
Water (check)		2769	55.4
L.S.D. .05		749	
.01		995	

*Mixture of the indicated metal salt of dimethyl-dithiocarbamic acid and 2-mercaptobenzothiazole.

These data (Table 1) show that under the conditions of this experiment, Karathane gave mildew control figures comparable with those obtained with sulfur. The degree of control obtained with Vancide F995W was not significantly different from that obtained with Karathane, but was less than with wettable sulfur. The apparent control obtained with Actidione at 1 p.p.m. and not at 2 p.p.m. is of doubtful significance. Extensive leaf injury obtained with this material made it difficult to estimate the area covered by the fungus. None of the other materials caused visible injury.

HORTICULTURAL CROPS RESEARCH BRANCH, AGRICULTURAL RESEARCH SERVICE,
U. S. DEPARTMENT OF AGRICULTURE

FACTORS INFLUENCING THE CONTROL OF PEACH SCAB IN SOUTH CAROLINA

Donald H. Petersen and John C. Dunegan[1]

The monographic study by Keitt[2] of the peach scab fungus, Cladosporium carpophilum Thuem., published in 1917, has long been considered the standard reference text on the subject. Keitt found that very few infections occur on the fruit until 4 to 6 weeks after petal fall. This finding confirmed the results obtained by earlier investigators and emphasized the importance of an application of a suitable fungicide about 1 month after petal fall.

His experiments also showed that scab could be controlled by spraying with self-boiled lime-sulfur, 16-16-100, or with wettable sulfur, 10 pounds in 100 gallons. Keitt considered it essential that all varieties receive a spray about 1 month after petal fall. If scab were the only problem, he felt that for maximum control a second spray should be applied 2 or 3 weeks later. However, the need in most orchards for protection against brown rot made it desirable to delay this second scab spray until 3 or 4 weeks before harvest. Varieties maturing later than Elberta need the spray applied 1 month after petal fall plus a second spray applied 2 or 3 weeks later plus a third spray 1 month after the second scab spray.

These control procedures have been known for many years. However, since 1951 peach growers in the southeastern fruit sections have reported each year a marked upsurge in the prevalence of peach scab on the fruit. It has been suggested that this increase might be due to the following:

(1) Absence of lime in the current spray formulations as the result of the use of organic insecticides.
(2) Reduction in the amount of fungicide used per application even though a larger total of fungicide is applied per season.
(3) Mechanical methods of applying sprays rather than the manual spraying formerly used.
(4) Possible development of a new strain of Cladosporium carpophilum.
(5) Increased susceptibility of the new peach varieties.

Our field observations and spraying experiments in South Carolina during 1953 did not furnish conclusive evidence that any single factor was responsible for the increase in the prevalence of peach scab. Accordingly, detailed plans were devised to investigate further in the 1954 experiments several of the factors involved.

EXPERIMENTAL PROCEDURE

The experimental program, involving 31 different treatments in 1954, was carried out in a block of 6-year-old Southland peach trees at Trenton, South Carolina. Single-tree plots, replicated 5 times, were sprayed manually with a hydraulic sprayer operating at 500 pounds per square inch at the pump. The trees were sprayed March 22 (petal fall), April 6 (shuck-split), April 15 (second cover) and April 26 (third cover). Preharvest sprays were applied May 18 and June 1.

All the trees (even those where the effect of lead arsenate was being tried) were treated with 1.5% parathion dust for the control of insects.

At harvest time 100 peaches were picked at random from each replicate tree and the number showing scab spots was recorded.

RESULTS

Effect of Time and Number of Sulfur Sprays on Control of Peach Scab: In this phase of the experiment, sulfur was used at the rate of 6 pounds per 100 gallons of water. The schedule of applications is shown diagramatically in Table 1, together with the amount of scab observed on the fruit examined at harvest time, and the results of a statistical analysis[3] of these data.

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1 Respectively, associate pathologist and principal pathologist, Horticultural Crops Research Branch, Agricultural Research Service, cooperating with Clemson Agricultural College.

2 Keitt, G. W., Peach scab and its control. U. S. Dept. Agr. Bul. 395, 1917.

3 E. J. Koch, of Biometrical Services, Agricultural Research Service, made the statistical analysis.

Table 1. Effect of time and number of sulfur sprays on control of peach scab.

Plot no.	Spray material and concentration	Spray applications[a]						Fruits with scab (Percent)	Mean equivalent angle
		Petal fall (March 22)	Shuck split (April 6)	Second cover (April 15)	Third cover (April 26)	Preharvest May 18	Preharvest June 1		
1	None (check)	---	---	---	---	---	---	98	82.95
2	Sulfur 6-100	XXX	---	---	---	---	---	98	84.11
3	"	---	XXX	---	---	---	---	97	81.49
4	"	---	---	XXX	---	---	---	96	79.00
5	"	---	---	---	XXX	---	---	47	43.16
6	"	---	---	---	---	XXX	---	87	69.27
7	"	---	---	---	---	---	XXX	91	72.33
8	"	---	---	XXX	---	XXX	---	66	54.66
9	"	---	---	XXX	---	XXX	XXX	47	43.04
10	"	---	---	XXX	XXX	XXX	---	8	15.82
11	"	---	---	XXX	XXX	XXX	XXX	11	18.58
12	"	---	XXX	XXX	XXX	---	---	16	23.43
13	"	XXX	---	XXX	XXX	XXX	XXX	13	21.09
14	"	XXX	XXX	---	XXX	XXX	XXX	5	12.64
15	"	XXX	XXX	XXX	---	XXX	XXX	27	30.48
16	"	XXX	XXX	XXX	XXX	---	XXX	16	22.92
17	"	XXX	XXX	XXX	XXX	XXX	---	10	18.15
18	"	XXX	XXX	XXX	XXX	XXX	XXX	2	7.45
L.S.D. 5%									6.48
1%									8.61

[a]XXX = indicated spray applied; --- spray not applied.

Under the procedure used (i.e., transformation of the percentage figures to equivalent angles) differences in significance must be measured from the means of equivalent angles and not from the arithmetical means.

The data in Table 1 show that a full-season schedule of six sulfur sprays (Plot 18) gave excellent control of scab. However, the omission of the second cover spray (Plot 14), or of the first two and the last applications (Plot 10), gave results that were practically as good.

The data show also that the spray applied April 26, 5 weeks after petal fall, had a marked effect upon the development of scab infections even when, as in Plot 5, this spray was the only one applied throughout the season. The higher incidence of scab infections in Plots, 8, 9, and 15 where the April 26 spray was omitted further emphasizes the importance of a spray applied during the period 4 to 6 weeks after petal fall.

The group of single-spray applications (Plots 2 to 7) are of considerable interest. The result obtained in Plot 5 has been mentioned, but it is evident that single applications on March 22, April 6, and April 15 (Plots 2, 3 and 4) respectively, were of little value in the control of scab, whereas the preharvest sprays (plots 6 and 7) did have a slight depressing effect upon the development of scab infections. It should not be inferred that any of these single-spray treatments are considered to give satisfactory commercial control.

The scab control obtained with the various combinations used in Plots 11, 12, 13, 16 and 17 is only slightly inferior to the control obtained in Plot 10. The results obtained in Plots 11, 12 and 13, where one or both of the early sprays were omitted, are as good as those obtained in Plots 16 and 17 where both early sprays were used, indicating that the early sprays have little effect upon peach scab control.

Effect on Scab Control of Various Chemicals Applied Only During the Scab Infection Period: The experiments just described involved the application of only sulfur at varied times. The next group of experiments tested the effectiveness of lime, lime and lead arsenate, lime and zinc sulfate, and lime, lead arsenate and zinc sulfate, applied only during the scab infection period. It was not feasible to have a treatment of only lead arsenate or zinc sulfate because of the drastic injury that would result. The results obtained with sulfur applied at the same time are included in Table 2 for comparison.

The data in Table 2 show that as compared with sulfur the other materials are not effective. The lack of control with lime alone (Plot 23) is particularly striking. The effect of the addition of lead arsenate to the lime (Plot 24) suggests, as has been maintained previously, that lead arsenate exerts some fungicidal action.

Effect of Materials Added to Sulfur Sprays on Control of Peach Scab: In this group of treatments a full season schedule (6 sprays) of sulfur was supplemented by certain applications of lime, of lime and lead arsenate, or of these two materials combined with zinc sulfate.

The results in Table 3 show that scab control was not markedly different in Plots 19, 20 and 21 from that obtained with the sulfur alone.

Plot 22, however, presents a different picture. Here the addition of zinc sulfate seemingly resulted in less satisfactory control. Direct comparison with the other treatments is not possible because the times the treatments were applied were not identical.

Effect of Increased Dosages of Sulfur on Peach Scab Control When the Critical (4 to 6 Weeks After Petal Fall) Spray is Omitted: The results that have been presented indicate the importance of the sulfur spray applied during the critical 4 to 6 weeks after petal fall period. In commercial practice, however, circumstances may arise (machinery breakdown or adverse weather conditions, for example) that affect the timing of this critical spray. The combinations shown in Table 4 were tested with such contingencies in mind.

Treatment 9 (sulfur at the rate of 6 pounds to 100 gallons of water) gave inferior control. When the amount of sulfur was increased to 8 pounds there was a marked increase in the control obtained and when 12 pounds of sulfur was used in each 100 gallons the control was equal to that obtained with a full schedule of six applications of sulfur at the rate of 6 pounds to 100 gallons.

Effectiveness of New Materials for Peach Scab Control: Endomycin, an antibiotic material reputed to have antifungal properties, and pyridimethione, an organic compound, were used in a full-season schedule of six applications. A sulfur-dichlone mixture was used in Plot 29 in the first three sprays followed by three applications of sulfur.

Endomycin and pyridimethione gave no indication of being useful for the control of peach scab (Table 5). Since the sulfur-dichlone mixture was used only in the three early sprays, its effect on scab control was not effectively tested in Plot 29.

Table 2. Effect on scab control of various chemicals applied only during the scab infection period.

Plot no.	Spray material and concentration	Spray applications[a]						Fruits with scab (Percent)
		Petal fall (March 22)	Shuck split (April 6)	Second cover (April 15)	Third cover (April 26)	Preharvest May 18	Preharvest June 1	
10	Sulfur 6-100	---	---	XXX	XXX	XXX	---	8
23	Lime 8-100	---	---	XXX	XXX	XXX	---	88
24	Lime 8-100 Lead arsenate 2-100	---	---	XXX	XXX	XXX	---	33
25	Lime 8-100 Zinc sulfate 4-100	---	---	XXX	XXX	XXX	---	51
26	Lime 8-100 Lead arsenate 2-100 Zinc sulfate 4-100	---	---	XXX	XXX	XXX	---	51

[a] XXX, indicated spray applied; ---, spray not applied.

Table 3. Effect of materials added to sulfur[a] sprays on control of peach scab.

Plot no.	Supplemental spray material and concentration	Spray applications[b]						Fruits with scab (Percent)
		Petal fall (March 22)	Shuck split (April 6)	Second cover (April 15)	Third cover (April 26)	Preharvest May 18	Preharvest June 1	
19	Lime 8-100	---	---	XXX	---	XXX	---	1
20	Lime 8-100 Lead arsenate 2-100	---	---	XXX	---	XXX	---	2
21	Lime 8-100 Lead arsenate 2-100 Zinc sulfate 4-100	---	---	XXX	---	XXX	---	5
22	Lime 8-100 Lead arsenate 2-100 Zinc sulfate 4-100	---	---	XXX	XXX	---	---	17

[a] All trees in Plots 19 to 22 received the full season schedule of 6 sulfur sprays in addition to the treatments listed in Column 2.

[b] XXX, indicated spray applied; ---, spray not applied.

Table 4. Effect of increased dosages of sulfur on peach scab control when the critical (4 to 6 weeks after petal fall) spray is omitted.

Plot No.	Spray material and concentration	Spray applications[a]						Fruits with scab (Percent)
		Petal fall (March 22)	Shuck split (April 6)	Second cover (April 15)	Third cover (April 26)	Preharvest May 18	Preharvest June 1	
9	Sulfur 6-100	---	---	XXX	---	XXX	XXX	47
30	Sulfur 8-100	---	---	XXX	---	XXX	XXX	19
31	Sulfur 12-100	---	---	XXX	---	XXX	XXX	1

[a] XXX, indicated spray applied; ---, spray not applied.

Table 5. Effectiveness of new materials for peach scab control.

Plot No.	Spray material and concentration	Spray applications[a]						Fruits with scab (Percent)
		Petal fall (March 22)	Shuck split (April 6)	Second cover (April 15)	Third cover (April 26)	Preharvest May 18	Preharvest June 1	
1	None (check)	---	---	---	---	---	---	98
27	Endomycin 100 p.p.m.	XXX	XXX	XXX	XXX	XXX	XXX	97
28	Pyridimethione 100 p.p.m.	XXX	XXX	XXX	XXX	XXX	XXX	95
29	Sulfur 6-100 Dichlone 4 ozs. - 100[b]	XXX	XXX	XXX	XXX	XXX	XXX	3
18	Sulfur 6-100	XXX	XXX	XXX	XXX	XXX	XXX	2

[a] XXX, indicated spray applied; ---, spray not applied.

[b] Dichlone not used in the last three sprays.

CONCLUSIONS

The results of one season's work rarely justify drawing any positive conclusions. However, the control of the peach scab has been no problem for many years. The results obtained in the various tests enumerated in this paper are in line with the general body of knowledge that has accumulated for many years. The following conclusions, therefore, can be drawn with considerable safety from the 1954 tests:

(1) The importance of a sulfur spray applied approximately 4 to 6 weeks after petal fall is re-demonstrated.
(2) Peach scab can be controlled with sprays of sulfur. It is not necessary to jeopardize the effectiveness of new organic insecticides by adding lime to the sulfur sprays.
(3) Lime used alone gave no indication of being of value for the control of peach scab.
(4) The addition of lead arsenate to lime enhanced the control of the scab. This mild fungicidal action of lead arsenate has been recognized for many years.
(5) The addition of zinc sulfate to the lead arsenate and lime mixture adversely affected the control of scab. However, a mixture of zinc sulfate and lime was superior to lime alone.
(6) If through unfavorable circumstances it is not possible to apply the critical 4 to 6 weeks spray, the results obtained in 1954 suggest that increasing the amount of sulfur from 6 to 12 pounds per 100 gallons of water will give satisfactory control. Environmental conditions fluctuate from season to season and it is conceivable that in some years conditions might be so favorable for scab development that even 12 pounds per 100 gallons would not give satisfactory peach scab control.
(7) Neither endomycin nor pyridimethione appeared promising for the control of peach scab.

HORTICULTURAL CROPS RESEARCH BRANCH, AGRICULTURAL RESEARCH SERVICE, U. S. DEPARTMENT OF AGRICULTURE, AND CLEMSON AGRICULTURAL COLLEGE, CLEMSON, SOUTH CAROLINA

FALL-DUG STRAWBERRY PLANTS AS SYMPTOMLESS CARRIERS OF THE RED STELE DISEASE

A. C. Goheen

Red stele is one of the most serious diseases of the strawberry throughout the Temperate Zones. In the United States, it may be serious as far south as eastern Virginia, Arkansas, and the coastal areas of California. In susceptible strawberry varieties, the disease may spread rapidly and soon render the entire planting worthless. Oospores of the red stele fungus, Phytophthora fragariae, contaminate the soil in fields where infected plants are grown and such fields cannot be replanted to strawberries for at least 13 years in some areas (2). The best method for controlling this disease appears to be preventing contamination of strawberry soils through the complete elimination of infected plants from strawberry planting stocks.

To provide planting stocks free from red stele, regulatory officials in the principal centers of strawberry-plant production inspect strawberry fields for signs and symptoms of the disease in early spring. If any trace of the disease is found, certification of the field is withheld. To be completely effective, this inspection must be made when macroscopic symptoms of the disease are apparent on the plants in the field.

Bain and Demaree (1) indicated that red stele can be diagnosed from macroscopic symptoms only during late winter and early spring. During fall and early winter, they found that incipient infections were present but because of the limited distribution and development of the characteristic root-rot symptoms the disease could be easily overlooked at that time. They also found that some light infections by the red stele fungus were impossible to diagnose from macroscopic symptoms even during late spring. It, therefore, appears that digging strawberry planting stocks during fall and early winter is exceedingly dangerous from the point of view of spreading red stele unless the complete histories of the soil in the field and of the planting stock used to set the field are known.

To ascertain whether plants that had been grown in soil known to be contaminated with spores of the red stele fungus would be free from infection if dug during the fall before symptoms of the disease were apparent on the plants we performed an experiment at Beltsville during the fall and winter of 1953-54. Plants of the Midland variety growing in contaminated soil in the spring of 1953 recovered from red stele when the soil temperatures rose to a level at which the red stele fungus was no longer active. During late spring and summer, a new bed of daughter plants was produced. On October 30, 44 plants were dug from this plant bed and the roots were thoroughly washed to free them from contaminating soil in so far as possible. No macroscopic evidence of red stele was apparent in the roots of either the first- or the second-year plants at this time. Approximately equal numbers of these washed first- and second-year plants were then set in steam-sterilized soil in an open coldframe. This soil was about 6 inches deep in a receptacle, or pan, made of heavy tarred paper in the bottom of which small holes were made for drainage. The tarred-paper receptacle was set into the sand on the bottom of the coldframe. Following rains, water stood at the bases of the plants for 1 or 2 days. The 1-year plants were set at one end of the receptacle, and the 2-year ones at the opposite end.

Hickman and English (3) demonstrated that high soil moisture content is necessary for development of red stele. Soil moisture during the early fall of 1953 was not favorable for the infection of strawberry roots by the red stele fungus. Although 2.79 inches of rain fell on October 28, 29, and 30, the 7-week period immediately prior was dry. The total precipitation from September 8 through October 27 was 0.75 inch. Consequently, the soil in the strawberry field was exceedingly dry just prior to setting up the above-described experiment. The soil temperature at the time the plants were taken was approximately 10° C.

On March 11, 1954, the plants in the tarred-paper receptacles were examined for macroscopic evidence of red stele. Of the 44 plants set, 43 had advanced root-rot symptoms of red stele. It is evident that at the time the plants were set, the red stele fungus was on or in their roots although no signs of infection were apparent.

This experiment shows that plants growing in red-stele-contaminated soil cannot be freed of the disease by digging the plants early in the fall and thoroughly washing the soil from the roots. Of more importance, it points to the danger of certifying fall-dug strawberry plants to be free from red stele. The plants that were set in the tarred-paper receptacle had no visible symptoms of red stele and on the basis of macroscopic examination they could have easily been certified as red-stele-free. Therefore, in any fall certification for freedom from red stele, the histories of the field in which the plants were grown and of the mother-plant stock used to set the fields must be considered.

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TOBACCO NECROSIS VIRUS ON STRAWBERRY

Norman W. Frazier

Summary

Tobacco necrosis virus was recovered from the roots of most greenhouse-grown strawberry plants tested, irrespective of whether the plants originated from seed or were propagated from established clones brought into the greenhouse. The virus was not recovered from foliage of infected plants nor were symptoms produced in inoculated strawberry leaves.

Tobacco necrosis virus was recovered from only 1 of 80 field-grown commercial or wild strawberry plants from 23 different localities. After growing in the greenhouse 4 months, 9 of the 79 virus-free field plants were retested and all proved to have become infected. The virus was recovered from roots of daughter plants on stolons developing freely on a moist redwood bench surface, but the virus was not detected in scrapings of bench-surface materials or in potting-mix components in absence of root tissue.

Data were obtained which indicated that: 1) tobacco necrosis virus was not systemically transmitted from infected mother plants to daughters through stolons; 2) the virus did not become systemic in the strawberry root system; 3) moist redwood bench surfaces and non-sterile clay pots were sources of tobacco necrosis virus contamination; and 4) infection of strawberry roots with tobacco necrosis virus did not appear to influence the expression of symptoms of strawberry mottle virus.

The association of tobacco necrosis virus with strawberry was first reported by J. P. Fulton (2) who recovered a tobacco necrosis virus from roots of Fragaria vesca (East Malling clone) and from other greenhouse-grown strawberries that had been grafted to plants of the East Malling clone. Fulton obtained no evidence to indicate that the potting mixture and tap water were sources of virus contamination, but his tests did give evidence of virus transmission when plants were runner-grafted to the East Malling clone. Only rarely could virus be recovered from non-grafted greenhouse-grown plants, and virus was not recovered from plants dug in commercial strawberry fields. He assumed that the virus had been introduced into the greenhouse in the F. vesca plants and interpreted his findings as indicating that the tobacco necrosis virus with which he worked was carried as a clonal contamination.

The present author likewise recovered tobacco necrosis virus from roots of greenhouse-grown strawberry plants, thus confirming the association reported by Fulton.

It appeared important to determine: 1) the extent to which the writer's greenhouse-grown strawberry virus indicator clones were contaminated with tobacco necrosis virus; 2) the sources of virus contamination; 3) whether the virus was carried systemically through stolons from mother to daughter plants; and 4) whether infection with tobacco necrosis virus influenced or altered the expression of symptoms of other strawberry viruses.

PROCEDURE

After initial tests of mechanical transmission of tobacco necrosis virus to Pinto beans and squash (varieties Bananna, Cocozelle, Bush Scallop, and Zucchini), Turkish tobacco was selected as the most satisfactory assay host. Mechanical inoculations were carried out by crushing tissue from inocula plants in an approximately 2-fold volume of 0.05M neutral potassium phosphate buffer solution and, without straining or pressing through cheesecloth, the crushed plant-buffer mixture was used up by gently rubbing it with a cotton swab onto 2 to 4 young matured leaves of a single tobacco plant previously dusted with 600-mesh carborundum powder. The inoculated plant was then immediately washed with a spray of tap water.

EXPERIMENTAL

Experiment 1. Tests were made for the presence of tobacco necrosis virus in greenhouse-grown strawberry virus indicator varieties of the F. vesca type and in wild and commercial strawberry varieties from the field.

Thirty-two greenhouse-grown plants were tested. From 12 plants, recoveries were attempted separately from both leaves and roots to Alpine (variety Baron von Solemaker?) strawberry, squash, Pinto bean, and Turkish tobacco. Typical local lesion symptoms of tobacco

necrosis virus were produced on bean, squash, and tobacco with the crushed root inoculum from 11 of the 12 strawberries. Tobacco necrosis virus was not recovered from leaf inoculum of any plant. No recognizable symptoms were produced on leaves of inoculated strawberry plants. It would appear in F. vesca type strawberries that tobacoo necrosis virus does not become systemic in leaves, nor are strawberry leaves susceptible to infection with the virus. Recoveries from the last 20 of the 32 strawberry plants were from roots to tobacco. Tobacco necrosis virus was recovered from each of the 20 plants. Thus, a total of 31 of 32 greenhouse-grown plants tested positive for the virus, a much higher incidence than previously reported by either J. P. Fulton (2) or R. W. Fulton (3). Among the 31 plants that tested positive were several kinds: 1) Alpine plants grown from seed, from which, in one instance, the virus was recovered from seedling plants still growing in a seed flat; 2) plants of clones that originated as hybrid seedlings from crosses made at Berkeley; and 3) plants of clones brought from outside sources into the greenhouse, including two wild California clones and the East Malling clone of F. vesca. Since nearly every plant tested was found to be contaminated and plants grown from seed were as heavily contaminated as those vegetatively propagated as clones, and, further, since clones that had originated within the greenhouse were as commonly infected as those brought in from the outside, the results gave no indication that the virus had been introduced into the greenhouse in contaminated clones or that its perpetuation in the greenhouse was associated in any way with clonal vegetative propagation. To the contrary, the evidence strongly indicated that a very potent source of tobacco necrosis virus existed within the greenhouse.

Tests were made of 49 wild F. californica plants growing in wooded areas in 13 California localities, 12 wild F. chiloensis plants growing along the ocean shore in 3 localities, and of 19 hybrid plants growing in 8 commercial strawberry fields, a total of 80 plants from 23 different localities. Only one of the plants, an F. californica found growing in a wooded suburb of Oakland and adjacent to a private garden, tested positive for tobacco necrosis virus. The results are similar to those of J. P. Fulton (2) who found strawberry plants from commercial fields to be consistently free of tobacco necrosis virus. The occurrence of tobacco necrosis virus on field plants under natural conditions was first reported from North America by R. W. Fulton (3) who found less than 0.1 percent natural infection in field-grown tulips at Madison, Wisconsin.

After the wild strawberry plants had been collected in the field and portions of their roots used in tests for tobacco necrosis virus they were potted and held in the greenhouse for further virus study. After 4 months, nine of the F. californica plants (tobacco necrosis virus-free when potted) were again tested and virus was recovered from each plant. These results further pointed to a source of tobacco necrosis virus within the greenhouse.

A limited number of tests were carried out in which small samples of the clay loam, peat moss, leaf mold, and garden compost used in the normal potting mix; rotting wood, moss, and soil film scraped from redwood greenhouse bench surfaces; and moss and dirt scraped from exterior surfaces of dirty clay pots in use, were mixed with buffer and rubbed on tobacco leaves. No tobacco necrosis virus was detected in any test.

Two tests were made from bare strawberry rootlets on daughter plants growing on stolons which were developing freely on a moist redwood bench surface. Both tests were positive for tobacco necrosis virus and indicated that the surface of the bench was a source of contamination although this was not detected in the inoculations using bench-surface materials in absence of root tissue.

Experiment 2. Since it appeared that testing by direct inoculation of potting-mix components was not a suitable method for detection of tobacco necrosis virus, strawberry plants were made use of in an experiment designed so that it might also provide evidence concerning systemic propagation of the virus.

Three plants of the East Malling F. vesca clone, from whose roots tobacco necrosis virus had previously been recovered, and on which stolons were beginning to develop, were selected as mother plants. Two stolons on two mother plants and three on the third were allowed to develop. As successive daughter plants were formed on each stolon, they were immediately pinned down to the surface of various test materials and allowed to become rooted. The daughter plants of all seven stolons were pinned to the materials in identical sequence. The materials tested included components normally used in the potting mix and several other materials which, by reason of the processes of their manufacture, should be tobacco necrosis virus-free. The materials, and the sequence in which the daughter plants were pinned in them, were: 1) complete potting mix; 2) vermiculite; 3) peat moss; 4) perlite; 5) oak leaf mold; 6) charcoal; 7) garden compost; 8) distilled water; and 9) complete potting mix.

tamination, the lower half of quart-sized parafined cardboard milk cartons were used. Small drainage slits were punched about 1 inch from the bottom of the cartons and the cartons were set directly on a redwood bench. All plants were watered with distilled water and fertilized with an inorganic fertilizer. Sixty-nine days elapsed between the dates when the first and last daughter plants were pinned. After variable periods of time the roots of all plants growing on single stolons were washed and tested for tobacco necrosis virus. Stolons were not broken between any of the plants until that time. The number of days that elapsed between pinning and testing of individual plants varied from 11 to 99.

Of 59 daughter plants, four tested positive for tobacco necrosis virus, as did all three mother plants that were retested with the daughters. All four positively testing daughter plants had been pinned in complete potting mix (three were 1st daughters and one a 9th daughter). It might be assumed that only the complete potting mix was contaminated with virus despite the fact that its individual components (garden compost, peat moss, and oak leaf mold) yielded negative results. The results also might be interpreted as indicating passage of virus through the stolon from the infected mother plants to the daughters. However, all four instances of tobacco necrosis virus recovery also could have been due to accidental contamination from the bench, and this appeared to be the most acceptable explanation. The cartons containing the three positively-testing 1st daughter plants had disintegrated so that root tips could have made contact with the bench surface. The 9th daughter had formed and rested on the bench surface for several days before it was pinned.

Of the planting materials tested, both root and leaf development were outstanding in the leaf mold and weakest in the peat moss and charcoal.

Experiment 3. Since the results of Experiment 2 were not conclusive, a further experiment was carried out to test the bench as a source of contamination.

Five plants of an F. californica hybrid indicator clone previously determined to be contaminated with tobacco necrosis virus were selected for mother plants, and two stolons were permitted to develop on four of the plants and three on the fifth. Half of the stolons on each plant were allowed to trail along a moist redwood bench surface, while the remainder were trained along the tops of milk cartons, which had been set on top of inverted, sterile, 5-inch clay pots in order to avoid bench contamination of the stolons. After the desired number of daughter plants had developed on the stolons, the milk cartons were filled with oak leaf mold and all daughter plants pinned down. In all, 13 daughters that had been protected from bench contact, and 16 that had trailed on the bench surface, were pinned. All plants were watered with tap water and sprayed with insecticides and fungicides and fertilized in the usual manner. After the daughters were pinned, the tips of all stolons were allowed to continue growth and trail on the bench surface. Following a growth period of 26, 31, 32, 33, and 39 days in the case of individual plants, tests for tobacco necrosis virus were made, a mother and all her daughter plants being tested simultaneously. In addition, four daughter plants were tested which had developed and become rooted on the bench after the proximal daughters had been pinned in the milk cartons.

The results of the tests were as follows: all 5 mother plants were positive; all 13 daughters on the stolons protected from bench contamination were negative; 1 of 16 daughters that had developed on the bench surface before pinning tested positive, 15 tested negative; 3 of the 4 bench-rooted plants tested positive. It appeared improbable that there had been any passage of virus systemically from infected mother plants through stolons to the roots of daughter plants. All instances of tobacco necrosis virus infection seemed most likely to have been due to contamination by contact with the bench surface.

The incidence of initial tobacco necrosis virus contamination among the 16 plants allowed to root on the bench before being pinned in leaf mold might be assumed to be equal to that of the group of four bench-rooted plants, but after approximately 5 weeks of growth in the cartons only one of the 16 plants gave evidence of infection. This could indicate that a certain degree of recovery or de-contamination took place. When the plants were pinned, the roots had developed usually less than 2 inches, and after pinning it is probable that entirely new roots were developed and the old ones died. In any event, after pinning root development was rapid, and the virus evidently was unable to maintain itself in the new roots except in one plant. This could be interpreted as evidence that the virus does not become systemic in the strawberry root system, but rather that it remains localized as it does in leaves of many host plants.

Experiment 4. An additional test was carried out to augment the data from previous experiments as to the source of infection in strawberry roots.

A plant of an Alpine hybrid indicator clone from whose roots tobacco necrosis virus had previously been recovered was selected for a mother plant and five stolons were allowed to develop on it simultaneously. Stolon 1 trailed on a moist redwood bench surface until five daughters had been formed and had developed a small root system on the bench surface. Each plant was then tested for tobacco necrosis virus by removal of half the rootlets, and after testing it was pinned in leaf mold in a 4-inch sterile clay pot. The base of each pot was set in the top of a milk carton so that the roots could grow downward through the drainage hole and hang free within the carton, protected from bench contamination. After 44 days, tests for tobacco necrosis virus were again made. One set of tests used the portions of the roots hanging free below the pot drainage hole, while a second set tested the proximal 3 inches of roots growing from the crown. Two of the five plants tested immediately before they were pinned were positive for virus. All plants tested virus-free in the later two sets of tests. These results added to the evidence that the bench surface afforded a source of tobacco necrosis virus and that the virus does not become systemic in strawberry roots.

Three daughters developed on a lateral of stolon 1 and two daughters on stolon 2. These five daughters were never permitted contact with the bench during their development and were pinned in leaf mold in milk cartons set on top of inverted 5-inch clay pots. After 51 days, the roots of all five plants tested negative for tobacco necrosis virus. The results indicated that infection could be prevented by protecting roots against contact with contaminated bench surfaces. The results also provided negative evidence on the passage of virus through stolons from an infected mother to daughter plants.

Stolon 3, with two plants, and a lateral of stolon 4 bearing three plants, were likwise prevented from bench contact during development. The daughter plants were pinned in leaf mold in sterile 4-inch clay pots set in milk cartons to prevent bench contamination. After 48 days, the roots of all five plants tested virus-free. The results were similar to those of the tests that included stolon 2.

The five daughters on stolon 4 were treated in the same manner as those on stolon 3, except that dirty non-sterile clay pots were used. After 48 days the roots of four of the five plants tested positive for tobacco necrosis virus. This test demonstrated that non-sterile clay pots could be a source of contamination.

Stolon 5 was not permitted bench contact. After developing, five daughters were pinned in leaf mold in sterile 4-inch clay pots, as in the test with stolon 3, but the pots were placed directly on the bench surface and the roots were allowed to grow out through the drainage hole at the bottom of the pot. After 44 days, two tests for tobacco necrosis virus were made of the roots of each plant: in one test only the distal portion of the roots that had grown through and beyond the pot drainage hole were used, while in the second test only the proximal 3 inches of the roots near the crown were used. The exposed distal roots of three of the five plants tested positive for the virus whereas all five tests of the proximal part were negative. The results again indicated that the redwood bench surface provided a source of tobacco necrosis virus, and, further, they were interesting because virus was detected in the distal portion of the roots that actually made contact with the virus source, but not in the proximal portion. This again suggested that tobacco necrosis virus does not spread systemically throughout the root system of strawberries.

DISCUSSION

The experimental evidence indicated that both used, non-sterile pots and wood bench surfaces could afford a ready source of tobacco necrosis virus when contacted by strawberry roots, and the high incidence of infection found in the author's greenhouse plants may be explained on that basis.

In experiments 2, 3, and 4 one objective was to obtain data concerning the effect of tobacco necrosis virus infection on the expression of symptoms of other strawberry viruses. In experiment 2, one mother plant was infected with strawberry mottle virus, while in experiment 3, three mother plants were infected with an unnamed virus. In neither experiment did any daughter plants of an infected mother test positive for tobacco necrosis virus, hence no symptom data resulted except that the tobacco necrosis virus-free daughters expressed symptoms consistent with those of the mother plants. In experiment 4 the mother plant was infected with strawberry mottle virus. In this experiment, 10 plants, including the mother plant, tested positive for tobacco necrosis virus while 16 plants tested negative. All plants displayed similar symptoms of mottle virus, and there was no evidence that such symptoms were affected in any way by tobacco necrosis virus. It was impossible to discern any influence of tobacco necrosis virus on the

growth, development, or appearance of the plants.

The work reported herein was carried on during all months of the year with no noticeable effect on the results due to the influence of seasonal progression. This appears to be contrary to the experience of other workers (1, 2, 3, 4, 5) who reported that recovery of tobacco necrosis virus from roots of greenhouse-grown plants was more consistent during the winter than at other times of the year. Fulton (3) also found the virus much more consistently in roots of plants grown at temperatures below 70° F than in those grown at 80°. It is possible that the more consistent results in the present experiments may have been due to the mild climate that prevails at Berkeley, where the greenhouse temperatures did not often exceed 80° even during the warmest months.

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DEPARTMENT OF ENTOMOLOGY AND PARASITOLOGY, UNIVERSITY OF CALIFORNIA, BERKELEY

INFECTIVITY AND STABILITY OF EXTRACTS FROM TOPS AND ROOTS OF CUCUMBER PLANTS INFECTED WITH A LATENT VIRUS DISEASE OF CHERRY[1]

N. Tomlinson[2]

During the course of a preliminary investigation of a latent virus disease of cherry in a cucumber host, it was noticed that aqueous extracts from the roots of diseased cucumber plants appeared to possess greater infectivity than similar extracts prepared from the tops of the same plants. Further investigation has shown that root extracts are not only more infective than those from tops, but are also more stable to aging at different temperatures.

MATERIALS AND METHODS: The virus disease was transmitted to cucumber (var. National Pickling) by mechanical inoculation of the cotyledons, using the carborundum technique (5). Macerated cherry flower petals (3, 4) from a sweet cherry tree (variety Gold), shown to contain a latent ring spot virus by indexing on Prunus serrulata, variety Shirofugen, served as the inoculum. The symptoms of the disease in cucumber appear to be identical with those described by Boyle et al. (2).

Cucumber plants were grown in the greenhouse in soil previously partially sterilized by steaming. The plants were held in the dark for 48 hours prior to inoculation (1), after which they were placed under artificial light provided by a bank of 40 watt fluorescent lamps (G. E. Daylight) spaced 6 inches apart and supported 3 feet above the plants. This light was supplied for 16 hours a day. The temperature varied between 70° and 80° F.

Inocula were prepared by grinding roots or tops of plants, 11 to 14 days following their initial infection, in a previously chilled mortar with ice-cold distilled water. Centrifuged extracts were prepared by centrifuging the macerated material for 3 minutes at 7,000 r.p.m. in the high speed attachment of an M. S. E. medium centrifuge. The decanted supernatant solution served as inoculum. The macerated material was kept at a temperature as close to 2° C. as possible during this preparation.

Extracts to be heated were placed in small flasks held in a water bath. The temperature of these extracts reached 48° C. after 30 seconds and 52° C. a few seconds later, and was maintained at that point for the remainder of the heating period. At the desired intervals aliquots of the extract were pipetted into test tubes in an ice-water bath and inoculations were carried out within.

When roots were to be used in preparation of an inoculum, they were first thoroughly washed with distilled water. Inoculum prepared from roots of healthy plants grown under the same conditions as infected plants was not infective. The extracts from either tops or roots were slightly acid in reaction, with pH in the range of 6.0 to 6.7.

Infectivity of inocula following treatment was determined by inoculating six plants with the preparation from each treatment, then observing the plants for appearance of symptoms for a period of at least 28 days.

RESULTS: The effect of dilution with distilled water on the infectivity of extracts from the tops and roots of the same plants was tested. In each case infectivity was rapidly lost with increasing dilution, but the root inoculum remained infective at a four-fold greater dilution (1 in 63) than did that prepared from the tops of the same plants. The effect of aging at different temperatures on the infectivity of extracts from the tops or roots of the same plants was also examined. As would be expected, the centrifuged extract from tops remained infective much longer at 2° than at 22° C. However, while centrifuged root extracts remained infective after 4 hours at 22° C., top extracts lost infectivity in between 3 and 4 hours at 2°. In subsequent work it appeared that although the infectivity and stability of different extracts varied, the relationship between extracts from roots and tops remained the same. A centrifuged root extract has been held at 2° for 18 hours with little if any loss in infectivity. A portion of the same extract held at 22° was infective after aging for 4 hours, but not after 6 hours. Heating at 52° quickly rendered extracts non-infective. However, while top extracts were inactivated in less than 2 minutes by such treatment, root extracts remained infective for 5 minutes.

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[1] Contribution No. 273 of the Chemistry Division, Department of Agriculture, Ottawa, Canada.

[2] Present address: Pacific Fisheries Experimental Station, 898 Richards Street, Vancouver 2, British Columbia.

The results were related to the fresh weight of plant material. The dry weight of either top or root was found to be about 5 percent of its fresh weight. Hence the observed difference between the two sources of inoculum would be the same on either a fresh or dry weight basis. Two possible explanations of the differences suggest themselves. One is that there is a higher concentration of virus in the root extracts than in the top extracts; the other, that there is a higher concentration of virus-inhibiting or inactivating substances (6) in the top extracts. In either event it appears that extracts from the roots of infected cucumber plants may be more suitable than those from the tops for use as starting material in the purification of the virus concerned in this disease.

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CHEMISTRY DIVISION, DOMINION PLANT PATHOLOGY LABORATORY, SUMMERLAND, BRITISH COLUMBIA

AN ATTEMPT TO USE A COLOR REACTION TEST TO IDENTIFY VIRUS-DISEASED GRAPEVINES

Betsy Hall and Wm. B. Hewitt

Reports of certain chemical tests to distinguish virus diseases in stone fruit trees (2, 3, 4) prompted similar tests with virus-diseased grapevines. The tests were run with dormant grape canes, with the leaves of healthy grapevines, with leaves from vines known to have yellow mosaic and fanleaf, and also with the leaves of a large number of seedling grapevines.

Color Tests with Dormant Wood of Grape Canes

Pieces of dormant canes from disased and healthy vines were collected from corresponding vine positions. After trying a number of samples of the bark, phloem, and xylem of the various canes, entire cross sections about 1 mm thick were used for color reaction tests. Five cubic centimeters of the following reagent were used in a test tube for each color reaction test:

Solution #1 40.0 gm NaOH dissolved in 500 cc distilled water
Solution #2 0.3 gm $CuSO_4$+3.0 gm Na Citrate dissolved in 500 cc distilled water

These two solutions were mixed in equal proportions when used.

The cross sections covered with the reagent in the test tube were heated in a boiling water bath for 10 minutes, allowed to cool, and then visually checked for color. Colors from strong pink to dark red were considered positive; no color reactions, yellow, or green, were considered negative.

Cane specimens were taken from 34 normal vines of as many varieties, from 25 vines of 25 different varieties infected with yellow mosaic, and from 53 vines of 34 varieties infected with fanleaf. From these tests, 26 of the control vines tested negative and 8 were positive; 13 of the yellow mosaic-diseased vines tested negative and 12 were positive; and of the 53 fanleaf-diseased vines, 32 tested negative and 21 were positive.

Under the conditions of these tests, the color reaction was not reliable in distinguishing yellow mosaic or fanleaf-diseased grapevines from the controls.

Color Tests with Leaves of Grapevines

A number of preliminary tests were conducted to determine the size of the sample and the amount of reagent which would give a readable color. Disks from grape leaves were cut from the midvein section close to the petiolar attachment with a #7-8 cork borer. Four cubic centimeters of the previously described reagent were used in each sample test tube.

The leaf disks, covered by the reagent, were heated in test tubes in a boiling water bath for ten minutes, allowed to cool, and then visually checked for presence and intensity of color. The following number and color key were used and the results tabulated by number:

0 - yellow or green (negative reaction)
1 - dark red (garnet brown (5))
2 - red (tea) (English red (5))
3 - orange (orange rufous (5))
4 - light orange or red
5 - brownish (a mixture of 0 and 3 or 4)

Every leaf from entire lateral canes was tested to determine if a color reaction was correlated with position. "Healthy" vines and mosaic- and fanleaf-diseased vines of the varieties French Colombard and Mission were sampled and tested.

Leaves of "healthy" vines of French Colombard and Mission varied in their color reaction from 1 to 5. There was no constant relation between color test and leaf position on the cane. Similar samples of leaves from mosaic- and fanleaf-diseased vines also varied in their color reaction. The terminal, basal, and median leaves varied from 0 or no reaction, to 1, a very strong red. It was not possible to distinguish the diseased from "healthy" vines by comparing the color reaction charts prepared with the results of these tests. Leaves of the diseased plants

gave about as many negative tests as those from the "healthy" plants.

It is possible that even the "healthy", or certainly normal-appearing, vines used in these tests could have been carrying a latent or masked virus, and therefore could give a degree of positive reaction to the color test. It is reasonable to assume that seedling vines would be free of virus, or at least a percentage of them from different parents could be expected to be free. Leaves from seedling vines of a number of different parentages growing in the breeding plots of Dr. H. P. Olmo of the Viticulture Department here at Davis, California, were sampled.

Sample leaves were taken from 214 one-year, 343 two-year, and 359 six-year old seedling vines growing on their own roots. Two leaves were selected from each vine; one leaf was fully matured but not degenerescent; the second leaf was a "recently matured" leaf (1). These samples were collected in the morning and kept overnight at 4° C. No variations occurred between samples kept in this way and samples which were tested immediately after collection.

Of the 916 seedling vines sampled, the color reaction of each of the two leaves from each of 449 vines was the same: 176 tested 0, 248 of them gave tests 1-2, and 25 of them tested 3-4. Each leaf of the two-leaf samples from the other 467 vines differed in their color reaction: 38 split between 5 and 0, 66 split between 5 and 1-4, 74 between 0 and 3-4, 192 between 0 and 1-2, and 97 between classes 3-4 and 1-2.

Some of the same vines were resampled one week to four weeks later, using the same two types of leaves to determine if vines could be resampled to give the same color reaction. Of 204 vines in which both leaves had given the same color reaction in the original sampling, only 63 gave the same color test as before. Of the 420 vines from the lot in which leaves differed in their color reaction, there were only 13 vines which tested the same as before,

To recheck again this variability of sample, 5 recently matured leaves were taken from each of 55 of the vines which had given identical color reactions in the two previous samplings. Only 9 of these vines gave the same color reactions as before in each of the 5 leaf samples.

Rather wide color variations resulted with this chemical test using dormant wood of grape canes, grape leaves from different positions, grape leaves with known virus, and grape leaves from seedling vines. Such widely divergent results lead to the conclusion that this chemical color test is not applicable to grapes. Problems in sampling may account for some of the variation.

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UNIVERSITY OF CALIFORNIA, DAVIS

SOME HOSTS AND VECTORS OF TOMATO ASPERMY VIRUS

Philip Brierley, Floyd F. Smith, and S. P. Doolittle[1]

Summary

Tomato aspermy virus, known in the United States since 1951, has been found to cause blight injury to flowers of some chrysanthemum varieties. Experimental inoculation resulted in infection in 38 species of 30 genera and 12 plant families and failed in 38 other species. No seed transmission was detected in China aster, tobacco, or Nicotiana glutinosa. The aphids Macrosiphoniella sanborni, Myzus persicae, and Myzus solani transmitted aspermy virus; Anuraphis helichrysi, Aphis gossypii, Macrosiphum solanifolii and Rhopalosiphum rufomaculatum failed to transmit it. Only M. solani transmitted the virus from chrysanthemum to chrysanthemum. Myzus persicae and M. solani acquired the aspermy virus in 15 minutes' feeding; both species lost infectiveness in 2 hours' post-acquisition starvation. On healthy test plants M. persicae retained the virus for 2 hours or less, and M. solani for 15 minutes or less. Aspermy and cucumber mosaic viruses are compared. Vector relations of aspermy virus are discussed in relation to field spread, which is thus far unreported in United States.

Following the detection of tomato aspermy virus in chrysanthemum in the United States in 1951 (3) we undertook to evaluate the potential importance of aspermy in this country. Although various European pathologists have found the aspermy virus associated with malformed blooms in chrysanthemum, Prentice (6) pointed out that the causal relation of aspermy virus to flower distortion is not established. He therefore proposed the name flower distortion for the disease characterized by severe dwarfing and malformation of chrysanthemum blooms. Brierley (2) recently reported the detection in the United States of a separate virus causing flower distortion in chrysanthemum similar to that described by Prentice and others. The aspermy virus, tested in parallel with this flower distortion virus, produced only slight dwarfing of blooms and irregular waviness of ray florets in the varieties Good News and Monument. Six other chrysanthemum varieties found naturally infected with aspermy virus expressed no flower symptoms, but healthy stocks of these varieties were not available for direct comparison. There is thus far little reason to regard the aspermy virus as a menace to American chrysanthemums, but it is known that this virus can damage the flowers of some varieties.

However, aspermy virus, originally described (1) as the cause of a disease of tomato, is capable of infecting other plants. Blencowe and Caldwell (1) reported natural infection in chrysanthemum and tomato, experimental infection in tobacco and Nicotiana glutinosa, and transmission by Myzus persicae. K. M. Smith (8) reported experimental infection of nasturtium, Phlox drummondii, zinnia, Helichrysum, wallflower, Primula sinensis var. stellata, lettuce, and soy bean with "the chrysanthemum mosaic virus", apparently the virus of tomato aspermy. Noordam (5) made an intensive study of "Cucumis virus 1, strain Chrysanthemum," which appears to be synonymous with tomato aspermy virus. He succeeded in infecting Antirrhinum maximum album, Callistephus sinensis, chrysanthemum, Cucumis sativus (local lesions only), Datura stramonium, Helianthus annuus, Helichrysum bracteatum monstrosum, Nicotiana glutinosa, N. paniculata, N. rustica, N. silvestris, N. tabacum, Petunia hybrida nana compacta, Primula obconica, Senecio cruentus grandiflora, Solanum lycopersicum (Lycopersicon esculentum), Tropaeolum majus nanum, and Zinnia elegans dahliiflora.

METHODS

Plants tested for susceptibility to aspermy were generally grown from seed and inoculated while young. Exceptions were chrysanthemum, Paris daisy, Chrysanthemum leucanthemum, Easter lily, coleus, dahlia, carnation, gladiolus, Saintpaulia, German ivy, potato, and Sparaxis, which were propagated vegetatively and inoculated during active growth.

Aspermy virus used in these experiments was isolated from the chrysanthemum variety Nightingale supplied to us by a firm in Ohio, which in turn received it from a California grower. Other cultures of this virus, isolated from other chrysanthemum varieties and originating in

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[1] Respectively, Pathologist, Horticultural Crops Research Branch, Entomologist, Entomology Research Branch, and Pathologist, Horticultural Crops Research Branch, Agricultural Research Service, United States Department of Agriculture, Beltsville, Maryland.

other States, produced essentially the same symptoms in tobacco and tomato. No evidence of important strain differences appeared in our experiments.

Tobacco or Physalis angulata plants served as sources of virus. The usual method of sap inoculation with the aid of carborundum was used. Groups of species tested at one time always included known susceptible species as a check on conditions of infection. Systemic or local infection and failure to infect were determined by subinoculation to tobacco, Physalis, or petunia by sap inoculation. Potatoes of two seedling lines were inoculated by grafting with scions of tobacco affected with aspermy. The aspermy virus was not recovered from potato by sap inoculation or by back-grafting potato scions on healthy tobacco 2 months after inoculation.

In insect-transmission trials from Nightingale chrysanthemum to tobacco and tomato, direct reading of symptoms was possible. In transfers to chrysanthemum Good News, sub-inoculation to tobacco, Physalis, or petunia was necessary to detect aspermy virus, which rarely produces recognizable symptoms in chrysanthemum leaves. Mottle symptoms in chrysanthemum, ascribed to the aspermy virus in our earlier report (4), are produced by Noordam's B virus, which is present together with aspermy virus in the Nightingale source variety.

In the insect-transmission trials, colonies of the leaf-curling plum aphid, Anuraphis helichrysi (Kltb.), the melon aphid, Aphis gossypii Glover, the black chrysanthemum aphid, Macrosiphoniella sanborni (Gill.), and the green chrysanthemum aphid, Rhopalosiphum rufomaculatum (Wilson), were established on source chrysanthemum plants of the variety Nightingale and transferred by brush to caged healthy chrysanthemum plants of the variety Good News, to seedling Marglobe tomato, or to seedling tobacco plants. In certain tests these aphids were permitted to move at will from heavily infested source plants to healthy ones in a community cage.

In the trials with the potato aphid, Macrosiphum solanifolii (Ashm.), from potato, the green peach aphid, Myzus persicae (Sulz.), from cabbage, and the foxglove aphid, M. solani (Kltb.), from potato or China aster, aphids were transferred to small covered glass dishes by brush and starved for 2 hours. Small pieces of leaves from source plants were then placed in the dishes and the aphids were allowed to feed on them for 15 minutes. On an average 25 aphids were then brushed onto the terminal leaves of each test plant and allowed to feed for 24 or 48 hours. At the end of this time the aphids were killed by immersing the plants in nicotine sulfate and soap solution.

In the trials to determine the persistency of the virus in the insect vectors, 15 aphids were transferred from the source leaf tissue to each plant in the first series, and the same aphids were then moved to another group of plants in the succeeding series.

RESULTS

Experimental Host Range: The following plant species or varieties were found susceptible to aspermy virus.

AIZOACEAE -- Tetragona expansa Murr., New Zealand spinach (local lesions).

AMARANTACEAE --Celosia argentea var. cristata (L.) Kuntze (local lesions).

BALSAMINACEAE -- Impatiens balsamina L. (local lesions).

CHENOPODIACEAE -- Beta vulgaris L., beet (local lesions); Spinacia oleracea L., spinach.

COMPOSITAE -- Calendula officinalis L.; Callistephus chinensis (L.) Nees, China aster; Chrysanthemum frutescens L., Paris daisy; C. leucanthemum L., whiteweed; C. morifolium (Ramat.) Hemsl., chrysanthemum; C. parthenium f. flosculosum (DC.) Beck, Matricaria Golden Ball; Emilia sagittata (Vahl) DC.; Gaillardia pulchella var. picta (Sweet) Gray; Galinsoga parviflora Cav.; Helichrysum bracteatum Andr.; Lactuca sativa L., lettuce; Senecio cruentus DC., cineraria; Verbesina encelioides (Cav.) B. & H.; Zinnia elegans Jacq.

CONVOLVULACEAE -- Convolvulus tricolor L. (local lesions); Ipomoea purpurea (L.) Roth (local lesions).

CUCURBITACEAE -- Cucumis sativus L., cucumber (local lesions).

LILIACEAE -- Lilium longiflorum Thunb., Easter lily.

POLEMONIACEAE -- Phlox drummondii Hook. (local lesions).

RANUNCULACEAE -- Delphinium ajacis L., larkspur.

SCROPHULARIACEAE -- Antirrhinum majus L., snapdragon.

SOLANACEAE -- Capsicum frutescens L., pepper; Datura innoxia Mill.; D. stramonium L.; Lycopersicon esculentum Mill., tomato; Nicotiana glutinosa L.; N. rustica L.; N. sylvestris Speg. & Comes (local lesions); N. tabacum L., tobacco; Petunia hybrida Vilm., petunia; Physalis angulata L.; P. floridana Rydb.; Solanum melongena L., eggplant (local lesions).

Insusceptible Plants: The following plants were found insusceptible to aspermy virus in parallel trials. Ambrosia artemisiifolia L., ragweed; Apium graveolens L., celery; Belamcanda chinensis (L.) DC., blackberry lily; Brassica oleracea var. capitata L., cabbage; B. oleracea var. botrytis L., broccoli; Cheiranthus cheiri L., wallflower; Coleus blumei Benth., coleus; Convolvulus sepium L.; Coreopsis douglasii (DC.) Hall; Cosmos sulphureus Cav.; Dahlia pinnata Cav., dahlia; Delphinium hybridum Hort., perennial delphinium; Dianthus caryophyllus L., carnation; D. chinensis L., annual pink; Erigeron annuus (L.) Pers., fleabane; E. canadensis L., fleabane; Fuchsia magellanica Lam., fuchsia; Gladiolus sp., gladiolus; Gossypium hirsutum L., cotton; Hemerocallis fulva L., daylily; Lilium formosanum (Baker) Wallace; Lychnis viscaria L.; Matthiola incana (L.) R. Br., stock; Phaseolus vulgaris L., bean, vars., Top Crop, Pinto, Primula polyantha Mill., primrose; Quamoclit sloteri Nieuwl., cardinal climber; Saintpaulia ionantha Wendl., African violet; Salvia splendens Ker, salvia; Sanvitalia procumbens Lam.; Senecio mikanioides Otto, German ivy; Silene compacta Fisch.; Solanum tuberosum L., potato; Sparaxis tricolor Ker; Tagetes erecta L., marigold; Tithonia rotundifolia (Mill.))Blake; Tropaeolum majus L., nasturtium; Vinca rosea L., periwinkle; Viola tricolor L., pansy; Zea mays L., corn.

Seed Transmission Trials: No evidence of seed transmission of aspermy virus was detected when 130 seedlings of Nicotiana glutinosa, 112 seedlings of tobacco, and 28 seedlings of China aster were grown from seed of infected plants.

Insect Transmission: Of the seven species of aphids tested as vectors of tomato aspermy virus, three species transmitted the disease (Table 1). Macrosiphoniella sanborni transmitted the virus from chrysanthemum to tobacco. Rhopalosiphum rufomaculatum failed to transmit. These two species are limited to chrysanthemum, their natural host plant; but in these tests they were observed to feed and survive for 4 days on tobacco plants. Myzus persicae transmitted the virus from chrysanthemum to tobacco and tomato, and also from tobacco and tomato to tomato. Myzus solani was the only species found to transmit from chrysanthemum to chrysanthemum and also the most efficient of the three vectors in the transfer of the virus from chrysanthemum to tobacco or from tobacco to tobacco and tomato. In tests with this aphid the inoculated tobacco and tomato plants showed characteristic bright-yellow mottling of young leaves, constriction of chlorotic areas, and downward bending of leaf petioles within 4 days after exposure to aphids. Leaves that developed later were normal on healthy plants or showed the typical tomato aspermy symptoms on infected plants. The transmission of tomato aspermy virus by M. solani, in spite of the direct injury that it causes by feeding on its host plants, is in contrast to the failure of this species to transmit other viruses in our tests.

In the present experiments the non-vectors included Anuraphis helichrysi, which causes marked chlorotic spotting and distortion of young leaves of chrysanthemum by direct feeding. Macrosiphum solanifolii and Aphis gossypii also failed to transmit the virus. Two strains of A. gossypii were tested; one of these feeds by preference on the older leaves of chrysanthemum and the other colonizes the young terminal leaves and stems.

The tomato aspermy virus is evidently of the non-persistent type (Table 2). Myzus persicae lost its ability to transmit within 2 hours and M. solani lost it within 15 minutes while feeding on healthy plants after removal from the source plants. Also, both species lost the virus within 2 hours of post-acquisition starvation.

DISCUSSION

We failed to infect wallflower (Cheiranthus cheiri) and nasturtium (Tropaeolum majus), even though previous workers had succeeded. Cucumber usually expressed no symptoms, but occasionally developed local lesions in cotyledons as reported by others. Annual delphinium (larkspur) was infected, but perennial delphinium was not. Lettuce var. Slobolt was severely dwarfed, but expressed no other symptom. Easter lily var. Creole, which carries a mottle virus of the tulip breaking group, expressed no additional symptoms following inoculation; aspermy virus was recovered 1 month after inoculation; but not from new shoots produced from the same bulbs 14 months later.

The failure of aspermy virus to infect celery, dahlia, perennial delphinium, gladiolus, Lilium formosanum, Sparaxis, pansy, and corn, which are known suscepts of cucumber mosaic virus; failure to induce necrotic fleck in Creole Easter lily as strains of cucumber mosaic virus regularly do; failure to induce systemic symptoms in cucumber; and infection of chrysanthemum and mosaic-resistant spinach are characteristics which contrast with those of

Table 1. Aphid transmission of tomato aspermy virus.

Insect species	Virus source plant	Plant inoculated	Number plants infected/exposed
Anuraphis helichrysi	Chrysanthemum	Good News chrysanthemum	0/10
Aphis gossypii	Chrysanthemum	China aster	0/4
		Chrysanthemum	0/23
		Tobacco	0/20
		Tomato	0/10
		Verbesina	0/10
Macrosiphoniella sanborni	Chrysanthemum	Chrysanthemum	0/20
		Tobacco	2/10
Macrosiphum solanifolii	Chrysanthemum	Tomato	0/5
		Tobacco	0/5
Myzus persicae	Chrysanthemum	Chrysanthemum	0/10
		Tobacco	16/45
		Tomato	5/20
	Tobacco	Tomato	5/10
	Tomato	Tomato	6/10
Myzus solani	Chrysanthemum	Chrysanthemum	+[a]
		Tobacco	5/5
	Tobacco	Tobacco	3/5
		Tomato	20/20
Rhopalosiphum rufomaculatum	Chrysanthemum	Chrysanthemum	0/30
		Tobacco	0/10

[a] Aspermy virus detected on indexing, but number of plants infected not determined.

Table 2. Non-persistency of tomato aspermy virus in Myzus persicae and M. solani on tobacco.

Pre-source starvation period	Acquisition period	Post-acquisition starvation period	Period on inoculated plants	Number plants infected/exposed	
				persicae	solani
2 hours	15 min.	0	0-15 min.	2/5	3/5
			15-60 min.	1/5	0/5
			1-2 hours	1/5	0/5
			2-3 hours	0/5	---
			3-4 hours	0/5	---
			4-5 hours	0/5	---
2 hours	15 min.	2 hours	2-3 hours	0/5	0/5
		4 hours	4-5 hours	0/5	0/5

generally accepted strains of cucumber mosaic virus. Also, Aphis gossypii and Macrosiphum solanifolii, important vectors of the cucumber mosaic virus, failed in our tests to transmit aspermy virus. Finally, the aspermy virus failed to protect zinnia against superinfection by Price's indicator strain of the cucumber mosaic virus.

Noordam (5) classed aspermy virus ("Cucumis virus 1, strain Chrysanthemum") as a strain of cucumber mosaic virus (Cucumis virus 1 K. M. Smith) and presented good evidence for this view. He found this virus infectious at dilutions of 10^{-5} or 10^{-6} in expressed sap of tobacco leaves, but not beyond 10^{-3} in sap of chrysanthemum leaves. The aspermy virus was

inactivated in 10 minutes at 70° C., not at 65°. Expressed sap held at 23° to 25° remained infectious for 12 hours or less. Electron photographs showed round particles similar to those found for cucumber virus 1 by Sill et al. (7). Noordam found that Myzus persicae could transmit aspermy virus as it does the cucumber mosaic virus, during brief feeding periods on source and test plants. He found that Price's indicator strain of cucumber mosaic virus gave a positive agglutination reaction with antiserum of the aspermy virus. Finally, he reported that aspermy virus, Price's indicator strain, and a cucumber mosaic virus isolated in the Netherlands, each generally protected tobacco and Nicotiana glutinosa against superinfection by either of the other two viruses.

In technical classification of the aspermy virus, the several available criteria must be evaluated. This virus shows relationship to the cucumber mosaic virus in many characteristics, but it differs more widely from the latter in host range and vector relations than do strains previously assigned to the cucumber mosaic virus group. Regardless of technical placement of the virus, practical considerations seem to demand retention of the common name tomato aspermy virus, for this virus poses different practical problems from those presented by the cucumber mosaic virus. Aspermy virus is not damaging to celery, cucumber, or Easter lily, in all of which cucumber mosaic virus causes serious diseases. Aspermy is a chrysanthemum disease, but cucumber mosaic is not, at least in the United States. The aspermy virus can persist in perennial hosts such as chrysanthemums and can be transmitted by aphids to annual crops such as tomato, pepper, lettuce and spinach. However, no instance of such natural spread to annual crops has yet been detected in the United States, although aspermy virus has been here for at least 4 years and has been identified in chrysanthemum in six States. Tomato is the crop in which aspermy might be expected to appear, inasmuch as tomatoes and chrysanthemums are commonly grown side by side in gardens. Aspermy symptoms in tomato, although distinctive, might be confused with the symptoms of other virus diseases by observers not familiar with aspermy. On the other hand, the vector relations of aspermy virus, together with the relatively low concentration of the virus in chrysanthemum, may serve to restrict its spread from chrysanthemum to other crops. Anuraphis helichrysi and Aphis gossypii, commonly found on chrysanthemum, are not vectors according to our data. Macrosiphoniella sanborni, also common on chrysanthemum, normally confines its activity to this crop, although it can be forced to feed on tobacco long enough to transmit the aspermy virus. Myzus persicae and Myzus solani are general feeders and effective vectors of aspermy virus but are not commonly seen on chrysanthemum.

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HORTICULTURAL CROPS AND ENTOMOLOGY RESEARCH BRANCHES,
AGRICULTURAL RESEARCH SERVICE, BELTSVILLE, MARYLAND

EVIDENCE INDICATING PRESENCE OF THE RATOON STUNTING VIRUS DISEASE OF SUGAR CANE IN PUERTO RICO

G. W. Bruehl[1]

In recent years a disease of sugarcane, the ratoon stunting disease, characterized by no external symptoms other than a reduction in vigor has been reported from Australia[2] and the United States[3]. No visible pathogens have been associated with it. It is carried in diseased seed pieces and is easily transmitted by juice on cutting knives, but no evidence of insect or other vector transmission has been discovered. The fact that neither infected stalk cuttings nor the plants grown from them show symptoms makes it only too easy for such a malady to go through rigid quarantine without detection. Therefore trials were begun in February, 1953, to determine whether the disease is present in Puerto Rico.

Recommended[4] hot water treatments were given to canes showing internally discolored nodal tissues. For the most part the prolonged heating killed the canes and severely injured most of the survivors, although C. P. 1161 was notable in its ability to survice prolonged hot water treatments. In many instances only single plants survived, and truly quantitative data were not obtainable. However, the regularity with which surviving heat-treated plants out-performed untreated ones (Table 1), together with the occurrence of internal stalk symptoms associated with the presence of the disease, leads to the conclusion that the ratoon stunting disease is present in Puerto Rico.

Table 1. Effect on green weight of hot water treatment of Puerto Rico sugarcane plant material for long enough to destroy the virus of ratoon stunting disease.

Treatment and material	Relative green weight untreated/treated
1.5 hours at 54° C. --	
C.P. 807. Plant cane, 8 months	56
C.P. 807. The same stools, 1st ratoon, 7 months	45
1.25 hours at 52°-54° C. --	
1st ratoon, 4 months:	
C.P. 807	57
C.P. 1161	76
C.P. 28/60	55
C.P. 29/116	17
C.P. 34/120	19
2 hours at 50° C. --	
1st ratoon, 4 months:	
Co. 214	71
H. 32-8560	40
Unknown[a]	69

[a] An unidentified grass which may be Narenga porphyrocoma (Hance) Bor., clone Shantir Bahrni.

[1] Formerly, Field Crops Research Branch, Agricultural Research Service, United States Department of Agriculture, and Insular Experiment Station, Rio Piedras, Puerto Rico.

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Plant cane seed cuttings of C. P. 807, C. P. 29/116, and Co. 214, treated and untreated, were planted in another field in rows 20 feet long, 2 rows each. The rows of C. P. 807 from treated seed had 18 percent more canes at 5 months, C. P. 29/116, 35 percent more, and Co. 214, 42 percent more.

Assuming that this virus is not carried in true seeds, seedling canes not yet cut with a knife might well be virus-free. On that basis four seedlings were cut with a sterile knife, two seed-pieces being planted uninoculated and two pieces of each inoculated[5] with juice of Co. 214 and planted. The uninoculated clones outyielded the inoculated ones by 19 percent in 8-month plant cane, and 59 percent in 5-month first ratoon. Seed of the 8-month old plant cane was planted in another locality and a stalk count showed 21 percent more canes in those rows planted with uninoculated cuttings.

A field of third ratoon P. R. 905 at Corozal was growing poorly, and the internal nodal tissues were highly discolored. Seed planted at Rio Piedras, part treated 2 hours at 50° C., and part untreated, gave a 60 percent difference in favor of the treated row. Juice of this P. R. 905 was extracted and used as inoculum to treated C. P. 1165. A reduction of about 60 percent green weight was observed in 6-month old first ratoon of the inoculated C. P. 1165.

FIELD CROPS RESEARCH BRANCH, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE, AND INSULAR EXPERIMENT STATION, RIO PIEDRAS, PUERTO RICO

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5 Two or three deep holes were bored into each seed piece with a 3/16-inch cork borer. Each hole was then filled by an eye dropper with juice expressed with a food chopper from the internal tissues of cane stalks.

FIELD INFECTION EXPERIMENTS WITH DWARF BUNT OF WINTER WHEAT IN ONTARIO[1]

R. J. Baylis[2]

Abstract

Spores of T. brevifaciens collected in Ontario during 1953 were germinated on water agar. In preliminary tests about 60 percent of the spores germinated within 8 weeks under light and at cool temperature; optimum germination occurred at approximately 5° C under constant light. Germination seldom occurred in darkness.

In field tests on infested soil, early and late fall plantings were made in which spores of dwarf bunt were applied to the seed, in the open drill, and on the soil surface after seeding.

The infections recorded in July 1954 showed that the early seeded wheat developed a crop containing less than 10 percent of dwarf bunt, whereas the late seeding produced from 50 to 80 percent of the plants infected. Furthermore in the late seeding the spores broadcast on the soil substantially increased infection. Seed treatments were not effective against infection from spores in the soil.

The results suggest that weather conditions and soil moisture from the time of seeding until infection are more important than the actual seeding date.

Dwarf bunt was first reported in Ontario in 1952 (1, 2). It is now present in at least 16 counties in the western part of the Province and is most prevalent in the major wheat areas bordering on Lake Huron. In Ontario, where winter wheat is grown on approximately 700,000 acres, the incidence of dwarf bunt, even at the low levels estimated by field and seed surveys of this year and 1953, constitutes a major threat to growers of high quality wheat.

Spores of Tilletia brevifaciens G. W. Fischer from bunted heads collected in 1953 were germinated in preliminary tests on water agar at controlled temperatures between 0° and 15° C. Germination began after 4 to 5 weeks and it was found that daylight or artificial light of low intensity acted as a stimulus. Although maximum percentage of germination under these conditions was evident by 8 weeks, it seldom exceeded 60 percent. The optimum temperature for germination under constant artificial lighting was approximately 5° C. Germination in total darkness, when it occurred, never exceeded 10 percent at any temperature tested.

The preliminary tests justify the conclusion that the dwarf bunt spores require light and a relatively cool temperature for germination. These observations on water agar are closely comparable with the results obtained by Gassner (3), who germinated spores of dwarf bunt on a soil substrate within similar periods of exposure and under somewhat the same conditions of temperature and lighting.

In the fall of 1953 a field infection test was made on soil of heavy clay type in Bruce County near Lake Huron in a section of a field where infection by dwarf bunt had been estimated at between 5 and 10 percent in the 1953 crop of winter wheat. Bunt-free seed of Cornell 595 was planted on the infested soil and it was hoped that some estimate would be obtained of the incidence of dwarf bunt in the year following the first observed outbreak of the disease. In this connection, an early as well as a late planting was made for comparison of a possible influence on infection. At the same time, tests were made to measure the effect of applying spores to the seed before planting, or in the drill, or spreading them on the soil immediately after seeding. An extensive test of seed treatments was also made by using bunt fungicides at slightly higher than the rates recommended for control of common bunt. Six treatments were applied dry: Ceresan M (3.2% Hg), Bunt Cure, Bunt-no-more (40% H.C.B.), Tritisan (60% P.C.N.B.), and two experimental mixtures of 1:1 Agrox C (5% Hg) with 40% H.C.B.

All plots were randomized and replicated four times. The infections became apparent in June and counts were recorded in July as percentages of dwarf-bunted heads in the total number of heads counted in two 10-foot rows at the center of each plot.

From the data presented it will be seen that regardless of treatments only a low level of infection was obtained from all plots of the early seeding.

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[1] Contribution No. 1434 from the Botany and Plant Pathology Division, Science Service, Canada Department of Agriculture, Ottawa, Ontario.

[2] Associate Plant Pathologist.

Table 1. Percentage of dwarf-bunted heads in mature winter wheat obtained from early and late seedings of bunt-free, and treated seed, on naturally infested clay; and by the addition of spores at the time of seeding. Ontario experiments, 1953-54.

Date of seeding / Treatment	Soil infested with dwarf bunt; from 1953 winter wheat crop			
	No spores added (Soil check)	Spores dusted on seed	Spores dusted in seed drill	Spores broadcast on soil surface after seeding
	Percentage of dwarf bunt			
Early seeding 12/9/53				
Check (no treatment)	3.2	2.5	6.7	3.2
Seed treatments[a]	1.9		6.3	3.0
Late seeding 1/10/53				
Check (no treatment)	57.2	61.0	63.3	78.4
Seed treatments[a]	52.1		50.3	80.4

[a] Average percent obtained from seed treatments with 6 different bunt fungicides used at slightly higher than rates recommended for common bunt control.

In contrast to this situation, the later seedings produced a very high percentage of dwarf-bunted heads and severe dwarfing of the plants. The seed treatments did not prevent infection from the spores in the soil; and, as the degree of infection was similar for all tests, the average percentage of the six treatments is used for comparison. Dwarf-bunt inoculum mixed with the seed before sowing, or in the open drill, did not significantly increase infection. In the late sowing, however, the spores broadcast on the soil immediately after the seed was sown substantially increased infection in all plots so treated.

No conclusions are possible at this time, but the results suggest that the weather conditions prevailing between seeding and the infection of the wheat are more important than the actual seeding date. In the case of the first seeding, made September 12, which is normal for the Bruce County area, the seed bed was prepared under unusually dry conditions. Immediately after seeding, however, the frequent warm rains during the next 2 weeks brought the moisture content of the soil up to near saturation point. The second seeding on October 1 was therefore made on thoroughly moist soil.

It would appear that in the case of the first seeding, the weather prevaling after September 12 was optimum for germination and quick emergence of the wheat, but the spores in the soil had not reached a point where they could germinate in sufficient numbers to cause heavy infection. After the October 1 seeding, the weather was again dry, but emergence was slow and took 2 weeks or more to be completed. During this period there was in the soil a reserve of stored moisture apparently sufficient to promote adequate spore germination. In this respect, the results differ from the observations made by Böning et al. (4) who assumed that a dry fall promotes infection.

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BOTANY AND PLANT PATHOLOGY LABORATORY, SCIENCE SERVICE, OTTAWA, ONTARIO

PRELIMINARY REPORT ON CROSS INOCULATIONS WITH DWARF BUNT ON WHEAT AND GRASSES[1]

Jack P. Meiners[2]

The occurrence of dwarf bunt (Tilletia caries (DC.) Tul.)[3] in several forage grasses has been reported from Idaho (1) and Oregon (2, 3). These infestations occurred in areas, and more specifically, in fields, that had a history of dwarf bunt in winter wheat, an association which has aroused speculation concerning the possible relationship between dwarf bunt of wheat and of various grasses. This is a report on cross inoculation experiments that were made to clarify this point.

Three dwarf bunt collections were used. These were obtained in eastern Oregon in 1952 from tall oatgrass (Arrhenatherum elatius (L.) Presl), intermediate wheatgrass (Agropyron intermedium (Host) Beauv.), and Elgin wheat (Triticum aestivum L.). The smut-infected wheat was growing as volunteer plants in a heavily infested field of tall oatgrass. Cross inoculations were made on these three hosts with spores from each by exposing seedlings to germinating spores in Petri dishes held at 10° C. for 14 days.

Infection was obtained in Elgin wheat inoculated with spores from all three hosts (Table 1), while no infection was obtained in tall oatgrass or intermediate wheatgrass with any of the spore collections. Failure to obtain dwarf bunt in these grasses has prompted further inoculation studies, which are now in progress.

Table 1. Percentages of smut obtained in Elgin wheat when inoculated with spores of dwarf bunt from grasses and wheat.

Inoculum from	Number heads		Percent smut
	Total	Smutted heads	
Tall oatgrass	144	40	27.8
Intermediate wheatgrass	141	9	6.4
Volunteer Elgin wheat	176	75	42.6

In an additional experiment on methods of inoculation, Red Bobs wheat was inoculated with spores of dwarf bunt from intermediate wheatgrass. Infection was obtained in 13 of 17 rows and the average percentage of smutted heads was 9.1. Parallel inoculations with spores from Elgin wheat produced infection in 11 of 17 rows, with an average smut percentage of 6.7.

These inoculation experiments provide the first experimental evidence that wheat is susceptible to dwarf bunt from tall oatgrass and intermediate wheatgrass.

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[1] Cooperative investigations of the United States Department of Agriculture, Field Crops Research Branch, and the Idaho and Washington Agricultural Experiment Stations.

[2] Agent, Field Crops Research Branch, Agricultural Research Service, U. S. Department of Agriculture.

[3] Also designated in the literature as T. brevifaciens G. W. Fischer, T. contraversa Kuehn, T. tritici (Bjerk.) Winter, and T. tritici nanifica Wagner.

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FIELD CROPS RESEARCH BRANCH, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE, AND WASHINGTON AGRICULTURAL EXPERIMENT STATIONS, PULLMAN, WASHINGTON

WAS DWARF BUNT IN NEW YORK STATE IN THE EARLY 1920'S?

D. C. Tingey

Tyler[1] reported dwarf bunt in New York in 1945. How long before that time it was present in the State is not known. However, Faris[2] in 1924 reported results of a series of experiments with Tilletia tritici (T. caries) and T. laevis (T. foetida) collected in New York. His data on T. caries indicate strongly that he was dealing with the dwarf bunt organism.

Faris reported that T. caries was collected in July 1923 from the variety Dawson, near Geneva, New York. To test germination spores of the two species were sprinkled on moist earth in petri dishes and incubated in an ice box. T. foetida germinated well. In contrast, T. caries germinated rather feebly, which is characteristic of dwarf bunt.

Faris presents data on the effect of temperature on infection by the two species on two wheat varieties, one of which was Dawson, apparently susceptible, since the original inoculum of T. caries was obtained from that variety. His "Table 1" shows a low percentage of infection for "T. tritici" as compared with the relatively high infection from "T. laevis", particularly at 5° and 10°C. The results with "T. tritici" are the results to be expected from seed inoculation with the dwarf bunt organism.

In discussing the results with T. caries, Faris writes, "I am at a loss to explain these low infections with T. tritici as the inoculum was collected and handled exactly the same as the T. laevis... It should be noted, however, that the smutted heads of T. tritici collected in the field appeared to be less than half the height of the sound heads in the same field." This observation is additional evidence that he was dealing with dwarf bunt. He states further that the smutty plants he obtained in his studies were normal in height. This could have resulted from a slight mixture of other races. Such mixtures were nearly always found in field collections of dwarf bunt in the present writer's studies in Utah.

Faris investigated the effect of moisture and character of substrata, and of date of planting, upon infection, using the same varieties and inoculum from the same two collections as before. Again he obtained a low percentage of infection with T. caries, contrasted with a high infection rate with T. foetida.

In another series of tests, Faris used four sources of inoculum, two for each species. One collection of T. caries was obtained from Washington; the other was the original New York collection. Two spring wheat varieties and the winter variety Dawson were used. All were spring planted. Data in "Table 4" of his paper show a high percentage infection for all but the New York collection of "T. tritici", which was practically zero as would be expected if the form involved were dwarf bunt, because dwarf bunt seldom causes bunted heads and rarely, if ever, from spring planting. The few plants that were infected could easily have been from mixtures of other races.

On the other hand, the "T. tritici" from Washington, which was obviously not of the dwarf bunt type, gave a relatively high infection, particularly on Dawson.

Faris' data in his "Tables 5 and 6" correspond with his other results with the New York collection of T. caries.

One further study by Faris, involving six collections of each species used as inoculum on ten varieties of wheat, is of interest. Two of the collections of T. caries failed to give a high percentage of infection. Both were New York collections, and according to Faris were made in 1923 at the same time as the one used in the previous studies. The other T. caries samples, obviously not dwarf bunt, resulted in a high percentage of infection on most of the varieties.

Faris concluded that his entire season's results with the New York collections of "T. tritici" had been unsatisfactory. However, the behavior of these New York collections is exactly what could have happened if they had been of the dwarf bunt type.

UTAH AGRICULTURAL EXPERIMENT STATION, LOGAN, UTAH

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CORRELATION BETWEEN RESISTANCE TO STEWART'S LEAF BLIGHT AND NORTHERN LEAF BLIGHT IN CORN

Benjamin Koehler

The first requisite for a breeding project to improve disease resistance in plants is to have means, either natural or induced, for assuring a frequent annual occurrence of the disease in the breeding nursery. Such means have been established for the Northern leaf blight disease of corn caused by Helminthosporium turcicum (1).

Stewart's disease caused by Bacterium stewartii is, on the average, of more importance than Northern leaf blight in Illinois, but no satisfactory methods have been developed for assuring the occurrence of the disease in the breeding plot. There is one advantage over some others in working with this disease, in that the expected occurrence at any location in any year can be predicted to some extent before planting time. This is due to the fact that the occurrence is more dependent on winter temperatures together with prevalence in the previous season, than on weather conditions during the growing season. In the many years when winter temperatures in the Corn Belt are too cold for overwintering, no progress in selection for resistance can be made. The only direct gains that have been made in control of the disease by breeding is through the determination of resistance or susceptibility of already existing inbred lines.

At Urbana, Illinois, a natural epidemic of Northern leaf blight, free from Stewart's disease, occurred in 1951. Stewart's disease occurred abundantly with the near absence of Northern leaf blight in 1953 and 1954. This gave excellent opportunity for rating corn lines and crosses to these diseases independently, even though there was some interference from drought in the last two years. When both diseases occur severely simultaneously, the writer has found it nearly impossible to take data on them separately.

In a previous publication (2) it was shown that for 26 inbred lines, a significant correlation occurred between scores for Northern leaf blight in 1951 and Stewart's leaf blight in 1953. Seventeen of the inbreds tested for Stewart's leaf blight in 1953 also were rated for Northern leaf blight by Elliott and Jenkins (1). Using their average results taken in August of 1942 and 1944, the correlation between the two diseases was 0.535, which is significant.

Robert, Jenkins, and Findley (3) have reported ratings on hybrids for three leaf blight diseases. Taking their Table 3, which reports on 30 two- and three-way corn-belt crosses, the correlations between disease ratings are as follows: H. turcicum and B. stewartii 0.606**, H. maydis and B. stewartii 0.432*, and H. turcicum and H. maydis 0.357*. Two stars indicate significance exceeding the 1 percent level, one star, the 5 percent level. Their Table 4 gives ratings for 35 corn-belt double crosses, and the correlations for the same disease combinations as above are 0.408*, 0.598**, 0.451*. Their Tables 5 and 6 involve hybrids of Wis. 355 and Wis. 240 maturity. For these two tables all the correlations are very low and not significant.

It seems logical that more emphasis should be given to the correlations obtained from data in their Tables 3 and 4 than in 5 and 6. The data in the first two are the average of two replications while the latter are based on only one replication. In the second place, the disease readings for H. turcicum and B. stewartii were considerably higher in the earlier hybrids reported in Tables 5 and 6 than in the corn-belt hybrids. This may have increased the difficulty of making accurate readings for both diseases occurring simultaneously.

Further evidence that resistance to Northern leaf blight and Stewart's leaf blight may be closely related was obtained in 1954. Recently breeders and plant pathologists in the U. S. Department of Agriculture, using the outcrossing and backcrossing method with selection under artificial epidemics of the disease, have greatly improved the resistance of some popular corn-belt inbred lines to the Northern leaf blight disease. A number of these recovered inbred lines were kindly supplied by A. J. Ullstrup and M. T. Jenkins. These were included as part of a test of 70 inbred lines planted at Urbana, May 6 and May 18. There were three replications for each planting. As there were no apparent differences in results between the two plantings, the data for all six replications were averaged together. The scores for Stewart's leaf blight were made on a range of 0 to 5 and were based on disease lesions independently of any apparent drought damage. In every case (Table 1) where comparisons are made between the original inbred line, and the recovered lines bred for resistance to Northern leaf blight, the Stewart's leaf blight rating is considerably lower in the recovered lines. In the case of smut, on the other hand (Table 1), the data show no improvement after breeding for Northern leaf blight resistance.

Concurrent resistance to two entirely unrelated diseases would be something unusual, but appears to exist for the two diseases mentioned among corn-belt inbred lines and crosses on

Table 1. Stewart's leaf blight and smut incidence in inbred lines of corn. Comparisons of the Station strain of the original inbreds with recovered lines of these inbreds which have been bred for resistance to Helminthosporium turcicum leaf blight. Urbana, Illinois, 1954.

Inbred	Time to tasseling (days)	Smutty plants (percent)	Stewart's leaf blight (score)
Ill. Hy2 (original)	73	1	1.0
Ind. Hy recovered[a]	75	3	.3
CI. 42A (Hy recovered)[b]	76	2	0
CI. 42B (Hy recovered)[b]	75	4	0
CI. 42C (Hy recovered)[b]	75	3	.8
Ind. 38-11 (original)	76	7	2.3
Ind. 38-11 recovered[a]	77	10	.8
CI. 38A(38-11 recovered)[b]	83	29	.8
CI. 38B(38-11 recovered)[b]	79	11	.3
Iowa L317 (original)	77	1	.5
Ind. L317 recovered[a]	81	4	0
Kan. K64 (original)	80	2	3.2
Ind. K64 recovered[b]	77	1	0
CI. 64 (K64 recovered)[b]	79	4	0

[a] Unreleased lines obtained from A. J. Ullstrup, U. S. D. A. and Purdue University.
[b] Unreleased lines obtained from M. T. Jenkins, U. S. D. A.

which data are available. Among these, there were only a few that failed to show a positive correlation. The progress already made in obtaining resistance to both diseases while working for Northern leaf blight (Table 1) will no doubt be of great value for the Corn Belt, as a good method for obtaining resistance to Stewart's disease independently has not yet been devised.

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ILLINOIS AGRICULTURAL EXPERIMENT STATION, URBANA

CO-OPERATIVE SEED TREATMENT TRIALS -- 1954[1]

J. E. Machacek[2]

In 1954, 15 seed treatments were compared under experimental plot conditions at 17 stations in Canada and 8 stations in the United States. In addition, the various products were tested in the greenhouse for phytotoxicity, for the control of seed rot, and for the minimum storage period required by the treated seed to provide adequate control of surface-borne smut. The seed used was Thatcher wheat naturally contaminated by bunt (mostly Tilletia foetida (Wallr.) Liro), Vanguard oats artificially contaminated (1-200, by weight) with spores of mixed Ustilago avenae (Pers.) Rostr. and U. kolleri Wille, Sanalta barley naturally contaminated by U. hordei (Pers.) Lagerh. and U. nigra Tapke, and Marine flax in which about 30 percent of the seeds had been cracked during threshing. No test was made of cereal seed infested by the seedling blight and spot blotch fungus Helminthosporium sativum Pam., King, & Bakke as sufficient naturally infested seed was not available.

The new seed disinfectants in the co-operative trials were as follows:

Ceresan M-2X -- A double-strength Ceresan M for use in machines treating seed by the slurry method. Contains 6.5% mercury as ethyl mercury p-toluene sulfonanilide. Supplied by Dupont Company of Canada, Ltd., Montreal, Que.

Co-op. Hexa SD -- A non-wettable dust containing 50% hexachlorobenzene. Supplied by Interprovincial Co-operatives Ltd., Winnipeg, Man.

Co-op. Mercury Seed Dressing -- A wettable dust containing 5% mercury as a mixture of phenyl mercury acetate and ethyl mercury chloride. Supplied by Interprovincial Co-operatives Ltd., Winnipeg, Man.

Dupont 244 -- A fluid containing 2.44% mercury as a mixture of phenyl mercury acetate and ethyl mercury acetate. Supplied by Dupont Company of Canada, Ltd., Montreal, Que.

Dupont 364 -- A fluid containing 3.64% mercury as a mixture of phenyl mercury acetate and ethyl mercury acetate. Supplied by Dupont Company of Canada, Ltd., Montreal, Que.

Gallotox -- A fluid containing 4% mercury as phenyl mercury acetate. Supplied by Gallowhur Chemicals Canada, Ltd., Montreal, Que.

Leytosan AL (name given to experimental product by writer) -- A dust containing 1.5% mercury as phenyl mercury urea (?) plus 12.5% aldrin. Supplied by Leytosan (Canada) Ltd., Winnipeg, Man.

Mercal -- A non-wettable dust containing 2% mercury as a mixture of phenyl mercury acetate and ethyl mercury chloride, plus 40% aldrin. Supplied by Green Cross Insecticides, Montreal, Que.

Mer-sol -- A fluid containing 4% mercury as phenyl mercury ammonium acetate. Supplied by Stauffer Chemical Company, North Portalnd, Oreg.

No Bunt -- A non-wettable dust containing 40% hexachlorobenzene. Supplied by Chipman Chemical Co., Winnipeg, Man.

Panogen 15 -- A fluid containing 1% mercury as methyl mercury dicyandiamide. Supplied by Green Cross Insecticides, Montreal, Que.

Puraseed -- A dust containing 3.8% mercury as phenyl mercury formamide. Supplied by Gallowhur Chemicals Canada Ltd., Montreal, Que.

Vitrioline -- A non-wettable dust containing 45% hexachlorobenzene. Supplied by Schlaesing Frères & Cie., Marseilles, France.

The results obtained with the above products, with respect to the control of surface-borne smut in wheat, oats, and barley, and seed rot in flax, are summarized in Table 1.

An assessment of phytotoxicity was obtained by comparing the results of a test with seed that had been recently treated with the results obtained after the treated seed had been stored at room temperature, in closed glass jars, for 3 or 4 months (Table 2). With certain seed dressings such as the mercurials and those containing hexachlorocyclohexane, phytotoxicity was rec-

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[1] Contribution No. 1430 from the Botany and Plant Pathology Division, Science Service, Canada Department of Agriculture, Ottawa, Ontario.

[2] Senior Plant Pathologist, Plant Pathology Laboratory, Winnipeg, Manitoba.

Table 1. Summary of results from field trials in 1954.

Treatment	Dose (oz./bu.)		Smutty heads or panicles (%)[a]			Seed rot (%)[a]
	Cereals	Flax	Wheat	Oats	Barley	Flax
Control (dry seed)	0.0	0.0	14.5	5.3	8.8	59
Ceresan M (dust)	0.5	1.5	0.5	0.1	2.9	49
Ceresan M-2X (slurry)	0.5[b]	1.5[b]	0.4	Tr[d]	1.6	47
Co-op. Hexa (dust)	0.5	1.5	0.3	2.9	8.0	59
Co-op. Mercury (dust)	0.5	1.5	0.4	Tr.	2.1	49
Dupont 244 (fluid)	0.75	2.25	0.5	Tr.	2.6	44
Dupont 364 (fluid)	0.5	1.5	0.9	0.1	2.4	46
Gallotox (fluid)	0.75	2.25	0.2	Tr.	1.5	45
Leytosan AL (dust)	2.0	2.0	0.9	Tr.	1.7	53
Mercal (dust)	2.0[c]	5.0	1.0	Tr.	2.1	51
Mer-sol (fluid)	0.5	1.5	0.7	0.2	2.5	45
No Bunt (dust)	0.5	1.5	0.5	2.4	8.1	59
Panogen 15 (fluid)	0.75	1.5	0.2	Tr.	3.2	42
Puraseed (dust)	0.5	1.5	1.0	Tr.	2.2	50
Vitrioline (dust)	0.5	1.5	0.5	3.0	8.0	58
Least significant difference (5% point)			4.2	0.8	1.9	4

[a] Means of data from 11 stations for wheat, 15 stations for oats, 18 stations for barley, and 11 stations for flax.

[b] After proper dilution.

[c] 1.4 oz. per bu. for oats and barley.

[d] Tr. = less than 0.1%.

Table 2. Phytotoxicity of treatments tested in 1954, and the minimum period of storage required for complete control of smut.

Treatment	Phytotoxicity[a]				Minimum period of storage (days)[b]		
	Wheat	Oats	Barley	Flax	Wheat	Oats	Barley
Ceresan M	+	+	0	0	?	0	?
Ceresan M-2X	+	+	0	0	4-8	1	?
Co-op. Hexa	++	+	0	0	0	?	?
Co-op. Mercury	++	++	0	0	1-4	0	?
Dupont 244	0	+	0	0	?	1	?
Dupont 364	0	++	0	0	?	0	?
Gallotox	0	++	0	0	?	0	1-4
Leytosan AL	0	+	0	0	?	0	?
Mercal	+	++	0	0	?	0	1
Mer-sol	0	++	0	0	?	0	4-8
No Bunt	++	++	++	0	0	?	?
Panogen 15	0	0	0	0	4-8	0	?
Puraseed	0	+	0	0	?	1-4	?
Vitrioline	++	++	++	0	0	?	?

[a] Phytotoxicity: ++ = slight; + = trace; 0 = none.

[b] Storage period: Some smutty heads developed even when seed was stored for 8 days. An increase in dosage may be required.

ognizable through the occurrence of hypertrophied seedlings, but in other cases it was evident by reduced germination only. As can be seen in Table 2, the products tested in 1954 did but little harm to the seed.

The minimum period for which treated seed should be stored before sowing was determined by the method used in 1953. Portions of the treated seed were sown in the greenhouse immediately after treatment and again after various periods, which in 1954 were after 1, 4, and 8 days. The plants from the seed were grown until they had headed, at which time a count of smutty heads or panicles was possible. The storage period was considered adequate when no smut developed (Table 2).

The writer wishes to thank the following co-operators who assisted with the seed treatment trials of 1954: Dr. C. S. Reddy (Ames, Iowa), Mr. A. A. Guitard (Beaverlodge, Alberta), Mr. H. Rhoads (Bozeman, Montana), Mr. W. H. Johnston (Brandon, Manitoba), Dr. C. M. Nagel (Brookings, South Dakota), Mr. J. E. Campbell (Charlottetown, Prince Edward Island), Drs. A. W. Henry and L. E. Tyner (Edmonton, Alberta), Dr. W. E. Brentzel (Fargo, North Dakota), Dr. W. F. Crosier (Geneva, New York), Dr. S. G. Fushtey (Guelph, Ontario), Mr. A. L. D. Martin (Indian Head, Saskatchewan), Messrs. A. D. McFadden and E. C. Lowe (Lacombe, Alberta), Mr. J. S. Horricks (Lethbridge, Alberta), Dr. D. C. Arny (Madison, Wisconsin), Mr. H. R. Ballantyne (Melfort, Saskatchewan), Mr. W. J. Breakey (Morden, Manitoba), Dr. V. R. Wallen (Ottawa, Ontario), Dr. C. S. Holton and Mr. L. H. Purdy (Pullman, Washington), Dr. R. O. Lachance (Ste. Anne de la Pocatiere, Quebec), Mr. M. B. Moore (St. Paul, Minnesota), Dr. R. C. Russell (Saskatoon, Saskatchewan), Mr. A. G. Kusch (Scott, Saskatchewan), Mr. D. S. McBean (Swift Current, Saskatchewan), and Mr. H. A. H. Wallace (Winnipeg, Manitoba).

PLANT PATHOLOGY LABORATORY, WINNIPEG, MANITOBA

SOYBEAN DISEASES IN IOWA IN 1954[1]

John Dunleavy[2]

Fusarium blight (Fusarium sp.), Phyllosticta leaf spot (Phyllosticta sojicola), stem canker (Diaporthe phaseolorum var. caulivora), bacterial blight (Pseudomonas glycinea) and bacterial pustule (Xanthomonas phaseoli var. sojense) were diseases of importance on soybean in Iowa in 1954.

Fusarium blight was especially prevalent in north-central Iowa. Many fields in this area were examined in July and almost half of them had infected plants. The roots of these plants were discolored and the only above ground symptom was moderate stunting of the plants. In a few fields up to 10 percent of the plants had been killed during an earlier stage of development. The dead plants occurred at random in the rows and were rarely found in localized areas. Examination of the same area in August revealed that up to 8 percent of previously infected plants had been killed in many fields. At this time the dead plants occurred in groups from 1/2 to 3 feet in length within the rows. Dead plants were more prevalent in low areas but were found on well drained areas as well.

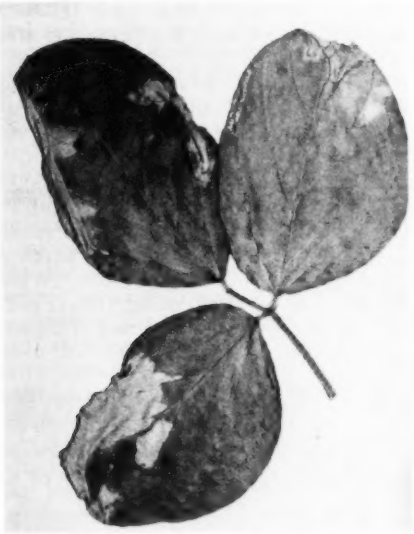

FIGURE 1. Phyllosticta leaf blight of soybean. Typical V-shaped necrotic area and faded color are evident on the leaflet at upper right.

Phyllosticta leaf blight affected almost all late-planted soybeans in Iowa that were planted after the first week in June. The most severe form of the disease as observed in both 1953 and 1954 was a leaf blight of seedlings. The unifoliate leaves were the first leaves to show symptoms, which developed first as a pale green V-shaped area that had its widest point at the margin of the leaf and became smaller as it penetrated the leaf tissue. Within 1 to 3 days, depending largely on drying conditions of the weather, the infected area of the leaf turned light brown. Infected areas usually began at the leaf margin near the tip of the leaf but lesions in the central portion of the leaf were not uncommon (Fig. 1).

The disease may spread to the first few trifoliate leaves but rarely develops beyond this point in the field. If plants are seriously infected prior to or at the time the third trifoliate leaves are unfolding tney appear to growers as dead or dying plants and may be disked into the soil. Frequently such fields are replanted to an earlier maturing variety of soybeans that becomes as seriously diseased as the first planting. Young plants may be defoliated, but no case has been observed in the past 2 growing seasons where plants have been killed as a result of

[1] Joint contribution from the Iowa Agricultural Experiment Station and the Field Crops Research Branch, Agricultural Research Service, U. S. Department of Agriculture. Jour. Paper No. J-2677 of the Iowa Agricultural Experiment Station, Ames, Iowa. Project No. 1179.

[2] Plant Pathologist.

Phyllosticta leaf blight. After the initial attack of the disease, plants recover rapidly by developing new leaves at the apical growing point and lateral branches which arise from buds. Disease damage to the crop appears to be a temporary slowing of normal plant development. Jehle et al. (1) have reported an outbreak of Phyllosticta canker and leaf spot in Maryland which affected more mature plants. During the latter portion of June and early July typical V-shaped Phyllosticta leaf spots were occasionally observed on more mature plants which had been planted in mid-May but this phase of the disease has been unimportant in Iowa. The varieties Blackhawk, Hawkeye, Adams, and Lincoln are all moderately susceptible to the disease. Certain lines of Glycine ussuriensis were very susceptible while others appeared very resistant. Seven lines of Glycine gracilis grown at Ames were all very susceptible. All the leaves on these plants were diseased throughout July and August.

Stem canker was not so prevalent as it has been the past few years. A few isolated fields were observed where the disease developed early in August, killing up to 6 percent of the plants. The percentage of plants infected in these fields ran as high as 43 in late September, but many of the plants included in this figure had already developed beans which were nearing maturity. Such late infection probably resulted in little loss in yield.

Bacterial blight was present in most soybean fields in northern and central Iowa throughout June and early July but in only a few isolated cases posed a serious disease problem. In the instances where bacterial blight was severe the disease was found exceptionally early. Heavy precipitation during June resulted in severe infection of the lower four to six trifoliate leaves which later were dropped. After the initial defoliation plants were not seriously affected by this disease.

Bacterial pustule, as in previous years, was widespread in southern Iowa. No defoliation of plants was observed due to this disease. About half the fields observed in late August and early September had from 50 to 97 percent of the leaves infected but the majority of leaves had only 1 to 5 percent of the leaf area involved.

Diseases of lesser importance were brown stem rot (Cephalosporium gregatum), downy mildew (Peronospora manshurica), bud blight (virus), and yellow bean mosaic (virus). Brown stem rot was much more prevalent than in recent years but occurred very late in the season. Affected fields had been planted primarily to the varieties Adams, Lincoln, and Clark which developed rotted stems just prior to maturity. Earlier maturing varieties were not affected except in fields where the disease was endemic. Downy mildew was most commonly observed on the susceptible variety Blackhawk. The majority of fields of this variety inspected in July had from 75 to 100 percent of the plants infected and an average of about 5 percent of the leaf area diseased. No defoliation was observed as a result of downy mildew.

There has been a considerable increase in yellow bean mosaic and bud blight in Iowa this year. In one field in southeastern Iowa a random sample showed 29 percent of the plants affected by yellow bean mosaic and 9 percent by bud blight. This field was unusual with respect to severity but, in general, it indicates the trend toward increased virus infection in areas where soybean viruses have not been a problem since about 1947. Although these two viruses were rarely observed in more than trace amounts, the number of fields in which they were observed has increased sharply.

Diseases of minor importance that were observed were brown spot (Septoria glycines), pod and stem blight (Diaporthe phaseolorum var. sojae), root and stem rot (Rhizoctonia solani), wildfire (Pseudomonas tabaci) and charcoal rot (Macrophomina phaseoli).

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DEPARTMENT OF BOTANY AND PLANT PATHOLOGY, IOWA STATE COLLEGE

AN OCCURRENCE OF MEALYBUGS AND OF BOTRYTIS ON SOYBEANS IN ONTARIO[1]

A. A. Hildebrand and H. R. Boyce[2]

Abstract

In August 1954, a species of mealybug, Pseudococcus maritimus (Ehrh.), was found on the roots of soybeans growing in the outdoor experimental plots at the Harrow Laboratory. Circumstantial evidence suggested infested roots of red clover growing adjacent to the soybeans as the probable source of the insects, and ants as the agents by which they were transported ffom the red clover to the soybeans. Contemporaneously, a representative of the genus Botrytis was found to be infecting the basal stem region of many soybean plants. Although neither insect nor fungus was causing any readily discernible injury to soybeans, nevertheless each is being investigated further, the former as a possible vector of the bud blight disease of soybeans caused by a virus of the tobacco ring spot group, the latter in its possible causal relationship to an apparently new disease that has commanded attention in Ontario this year.

In August of the current year two observations of interest and, perhaps, of some significance, were made in the soybean experimental plots at the Harrow laboratory. The first involved the discovery of mealybugs on the roots of the plants, the second the finding of a species of Botrytis at the base of the stem of many plants.

MEALYBUGS

Preparatory to the resumption of seasonal field studies of stem canker (Diaporthe) plants in the plots were selected for inclusion later in proposed pathogenicity trials. Unsuitable plants were pulled and discarded. During the course of these operations on August 4, mealybugs were noticed on the roots of a pulled plant. Further examination of the roots of a large number of plants of several varieties showed that many of them, regardless of variety, were more or less heavily infested with mealybugs in various stages of development (Fig. 1). Often, instead of the insects themselves, the roots showed adhering cottony wefts in which the eggs had been deposited (Fig. 2). In the section of the plots where the earlier root examinations were made, mealybug-infested plants occurred more or less haphazardly, and in no instance did above-ground parts of plants give an indication of the presence of the insects on their roots. Invariably, evidence of any activity at the base of the stem of a plant was a sure clue to the presence of mealybugs on the roots. The cornfield ant, Lasius alienus americanus Emery, was consistently associated with the presence of the mealybugs.

In 1953, the soybean experimental plots had occupied identically the same site as in 1954. In 1953, in the course of routine pathological studies, the roots of many soybean plants had been examined but mealybugs had never been observed. This fact suggests that the invasion by the insect must have been of more recent date. The obvious association of the ants with the mealybugs suggested also a probable means by which the latter had been spread from some other location. These circumstances led to an examination of the roots of weed and cultivated host plants in plots adjacent to the soybeans. Heavy populations of P. maritimus were found on the roots of red clover plants (Trifolium pratense L.) growing in a plot which bordered one end of the soybean plot and which was separated from the latter by an unplanted strip about 6 feet wide. Thus it appeared that the red clover may have been the source from which the mealybugs were transported to the soybeans. What strengthened this supposition was the fact that soybean plants in the rows immediately adjacent to the clover were more consistently infested than those farther away.

Prior to the discovery of the mealybugs, the weather had been extremely dry, and the non-cultivated red clover would, presumably, have suffered more severely from the drouth than the regularly cultivated soybeans. The greater succulence of the soybean roots may have incited

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[1] Joint contribution from the Botany and Plant Pathology Division (Contribution No. 1413) and the Entomology Division (Contribution No. 3267), Science Service, Canada Department of Agriculture, Ottawa, Ontario.

[2] Senior Plant Pathologist, and Entomologist, respectively.

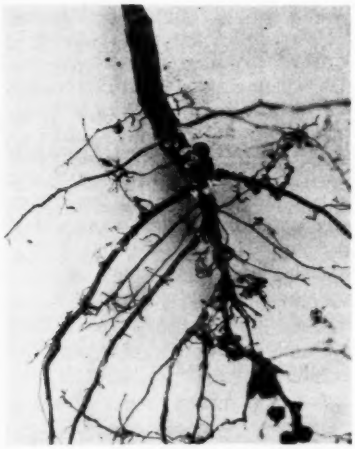

FIGURE 1. Root of soybean showing mealybugs in situ.

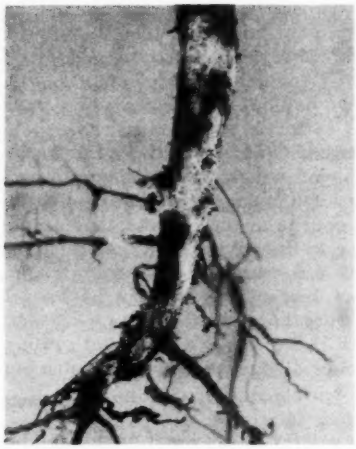

FIGURE 2. Root of soybean showing cottony wefts in which eggs of mealybugs were deposited.

the ants to transfer their protégés. Specimens of mealybugs from red clover and from soybeans were sent to the Entomology Division, Ottawa, where they were identified by W. R. Richards as Pseudococcus maritimus (Ehrh.), commonly known as the grape mealybug. There appear to be no Canadian records of this species from soybean or red clover. In 1941, McCulloch and Weigel (4) in the United States indicated that infestation of gladiolus corms by P. maritimus was greater when plantings were made in fields previously occupied by clover or weeds.

In 1951, Chamberlain (1), in summarizing soybean disease investigations in the North Central States of the United States, commented about bud blight caused by a virus of the tobacco ring spot group as follows: "In connection with this disease an interesting observation was made at Rosholt, South Dakota. In a soybean nursery adjacent to two small fields of red clover and alfalfa, 'dudding' was apparent in plants along the border rows. A short distance away, still contiguous to the soybean nursery, was a wheat field, adjacent to the forage legumes. Plants in the border rows opposite the wheat field showed no 'dudding'. This suggested an insect vector from the legumes and probably one of limited motility, such as larvae or early instars."

At Harrow this year, of the 903 soybean plants, variety Blackhawk, which consituted the population of the five rows bordering the clover plot, 156, or 17.2 percent, were found to be infected with bud blight. In a second five-row strip of the same variety, some 40 feet removed from the clover, only 14, or 1.6 percent of the 837 plants comprising the population of these rows, showed bud blight infection. Thus there was strong circumstantial evidence of a correlation between location of bud-blight-infected plants and the pattern of infestation by the mealybugs. The possibility of P. maritimus being a vector of bud blight is being further explored. Of related interest is the fact that Newton (5) has recently found Planococcus citri (Risso) capable of transmitting tobacco mosaic.

BOTRYTIS

On August 10, when plants were being inoculated and attention was necessarily focussed on the lower region of the stems, a grayish mould was observed on a number of plants. The mould, which enveloped the stems to a height of about 2 inches above the ground level with a more or less discrete and often moisture-laden mycelial growth, was not conspicuous and might easily have escaped detection at usual eye level. Examined microscopically, the fungus was found to be a representative of the genus Botrytis. As inoculations of a large number of plants of the varieties Blackhawk and Lincoln were proceeded with in August, mould-affected plants of both varieties continued to be found, though with noticeably diminishing frequency as

the end of the month approached. Although the incidence of a fungus like Botrytis might be expected to be correlated with the increased or prolonged humidity that may occur where growth or stand of plants is densest, the sporadic occurrence of infected plants throughout the plots regardless of density or vigor of growth suggested no such correlation. Examination of infected plants throughout the season revealed no really significant pathological effects. On September 3, 50 plants of the variety Lincoln were inoculated by the toothpick method (2) with a pure-culture isolate of the fungus. None of the inoculated plants showed any evidence of infection. The inoculations, it may be pointed out, were carried out after it was no longer possible to find natural infection in the field, and presumably, therefore, under conditions not favorable for the development of the fungus.

The above observations seem to discount almost entirely the importance of the Botrytis encountered as a pathogen of the soybean. There is, however, another circumstance which suggests that the possible significance of Botrytis should be investigated further. During the past summer a large number of diseased specimens of soybeans have been received at the Harrow laboratory from many sections of the soybean-growing areas of southwestern Ontario. From these plants, the symptoms of which were not recognized as typical of those of diseases encountered in the past, the fungus Botrytis has been isolated with a consistency that does not appear to be without significance.

As far as the writers are aware, there are only two references in the literature regarding Botrytis on soybeans. In 1922, Pape (6) reported from Germany that a species of Botrytis which had evidently first attacked young pods on the upper part of the plant later infected the main axis. In 1931, Lobik (3) reported that a species of Botrytis attacked a variety of soybeans in the North Caucasus region in Europe.

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SCIENCE SERVICE LABORATORY, HARROW, ONTARIO, CANADA

EFFECT OF PLANTING DATE ON INCIDENCE OF FUNGUS INFECTION OF OGDEN SOYBEAN SEEDS GROWN AT WALNUT HILL, FLORIDA

R. A. Kilpatrick and E. E. Hartwig[1]

Summary

Ogden soybean seeds were obtained from a date of planting study grown at Walnut Hill, Florida, in 1952 and 1953. Counts of seed from each date of planting showed the visible purple stain to be more prevalent in 1952 than in 1953. Early planted plots in 1952 showed more purple stain than later planted ones. Isolations from apparently healthy soybean seed yielded 13 different fungi. The purple seed stain fungus, Cercospora kikuchii, and Phomopsis sojae were most frequently isolated. C. kikuchii was more commonly isolated from seed of early plantings in 1952 and later plantings in 1953. Phomopsis sojae was isolated most frequently from seed of early plantings and only rarely from seed of late plantings in both years.

Seed samples of the Ogden variety of soybean (Glycine max (L.) Merrill) obtained from a date of planting test at Walnut Hill, Florida[2], in 1952 contained a high percentage of seeds stained purple by the fungus Cercospora kikuchii (T. Matsu. & Tomoyasu) Gardner. Appearance of the seed and the manifestation of purpling seemed to be influenced considerably by date of planting. Consequently, a detailed study of the seed was conducted to determine: 1) The relative prevalence of external (visible) and internal (non-visible) purple stain infection; and 2) the kind of fungi that could be isolated from seed of the various planting dates.

Table 1. Percentage of purple-stained seeds occurring on Ogden soybeans grown at Walnut Hill, Florida, in 1952 and 1953.

Date of planting	Percentage of seeds showing purple stain	
	1952	1953
April 15	35	3
May 1	37	4
May 15	18	2
June 1	8	3
June 15	4	4
July 1	4	2
July 15	-	4
August 1	0.4	7

One-pound samples were obtained from plots planted at biweekly intervals from April 15 to August 1. The percentage of purple stained seed was determined by actual count of discolored seed from all planting dates. In addition to making the visual examination for purple stain, 200 apparently healthy seeds from each planting date of the Ogden variety were plated to determine the percentage of internal infection by C. kikuchii. These seeds were surface sterilized in mercuric chloride (1:1000) for one minute, washed in sterile distilled water, and plated on potato dextrose agar containing 50 p.p.m. of the sodium salt of 2, 4-D acid (1). The petri plates containing five seeds each were incubated at room temperatures (75° to 95° F.) for 8 days. The fungi growing from infected seeds were identified.

In 1953, 1/4-pound seed samples of Roanoke, Jackson, and Lee were examined along with Ogden. Since the percentage of purple stain was much greater on the Ogden variety, only the

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[1] Plant Pathologist and Research Agronomist, respectively.

[2] Thanks are due to Mr. Ralph Smith, Agronomist, Mobile Unit #2 of the North Florida Experiment Station, Quincy, Florida, for furnishing the seed samples used in these tests.

Table 2. Relative prevalence of various fungi isolated from apparently healthy Ogden soybean seed obtained from a date of planting test at Walnut Hill, Florida, in 1952-53.

Fungus	Percent of total isolates[a]	
	1952	1953
Cercospora kikuchii	33	21
Phomopsis sojae	32	37
Colletorichum truncatum	9	Tr.
Aspergillus spp.	5	5
Aspergillus niger	5	1
Penicillium spp.	4	1
Cladosporium spp.	2	2
Fusarium oxysporum	1	17
Curvularia sp.	1	Tr.
Chaetomium sp.	0	12
Alternaria spp.	0	1
Helminthosporium spp.	0	Tr.
Rhizopus nigricans	0	Tr.
Unidentified spp.	8	2

[a] All planting dates combined.

Table 3. Relative prevalence of the two fungi isolated most frequently from apparently healthy Ogden soybean seeds obtained from a date of planting test grown at Walnut Hill, Florida, in 1952 and 1953.

Planting date	Percent of total isolates[a]			
	Cercospora kikuchii		Phomopsis sojae	
	1952	1953	1952	1953
April 15	26	4	45	78
May 1	39	7	31	65
May 15	43	14	35	47
June 1	30	21	35	26
June 15	38	42	13	20
July 1	15	21	0	12
July 15	-	17	-	11
August 1	18	60	0	0

[a] Based on isolations made from 200 apparently healthy seeds from each sample.

data from this variety are presented.

Seed from the earlier plantings in 1952 contained a much higher percentage of visibly purple-stained seed than those from later plantings (Table 1). This difference was not evident in 1953 when much less purple stain developed in the plots.

Fungi other than the purple seed stain organism were isolated each year from the apparently healthy seed. Phomopsis sojae was isolated readily from the seed each year, but the prevalence of other fungi varied considerably in both years (Table 2). Previous investigations (2, 3, 4) have shown the purple seed stain fungus can be isolated from a high percentage of apparently healthy soybean seeds.

The prevalence of the two fungi isolated most frequently is shown in Table 3 as percent of the total isolates obtained from the sample for each planting date. Cercospora kikuchii was isolated most frequently from seed harvested from May planted plots in 1952, but in 1953 this fungus was most prevalent in seed harvested from plots planted August 1. Phomopsis sojae, on the other hand, did not show this seasonal variability. It was isolated most frequently each year from seed harvested from the earlier planted plots (April 15 to June 15) and only occasionally from seed harvested from plots planted July 1 or later. The data suggest that certain weather conditions or host-environmental relationships are responsible for seasonal prevalence and visible expression of purple stain on soybean seeds.

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FIELD CROPS RESEARCH BRANCH, AGRICULTURAL RESEARCH SERVICE, U. S. DEPARTMENT OF AGRICULTURE, IN COOPERATION WITH THE DELTA BRANCH EXPERIMENT STATION, STONEVILLE, MISSISSIPPI

FUNGUS INFECTION OF SOYBEAN SEED AS INFLUENCED BY STINK BUG INJURY

R. A. Kilpatrick and E. E. Hartwig[1]

Summary

Soybean seeds obtained from a date of planting study at Walnut Hill, Florida, in 1952 and 1953, showed a high percentage of stinkbug-injured seeds, especially at the later planting dates. Isolations from punctured and non-punctured seed yielded a number of different fungi; however, the data suggest that stinkbug injury is not necessary for fungus infection of soybean seed. The symptoms on the seed varied, depending on stage of development when injury occurred. Seeds punctured at an early stage of development were small, shriveled and poorly developed. Seed injured at a later stage generally showed the puncture surrounded by a discolored ring.

Seed samples of the Ogden variety of soybean (Glycine max (L.) Merrill) obtained from a date of planting study at Walnut Hill, Florida[2], in 1952, contained a high percentage of seeds punctured by the southern green stinkbug, Nezara viridula (L.). A study was made to determine: 1) the fungi associated with stinkbug-injured seeds; and 2) the relationship of planting date to the prevalence of the injury.

Table 1. Relative prevalence of fungi isolated from stinkbug-injured seed of different varieties of soybeans.

Fungus	Percent of total isolates by varieties[a]			
	Ogden	Jackson	Roanoke	Lee
Cercospora kikuchii	47	27	34	35
Chaetomium sp.	13	35	23	26
Fusarium oxysporum	6	14	19	11
Cladosporium spp.	4	3	6	3
Aspergillus sp.	4	1	2	6
Alternaria spp.	2	3	6	4
Phomopsis sojae	1	5	-	5
Penicillium spp.	6	-	-	1
Fusarium roseum	3	-	2	1
Colletotrichum truncatum	-	3	-	2
Helminthosprium spp.	3	-	-	-
Phyllosticta sp.	1	-	-	-
Trichoderma sp.	1	1	-	-
Colletotrichum destructivum	-	2	-	-
Aspergillus niger	-	-	2	-
Rhizopus nigricans	-	-	-	1
Curvularia sp.	1	-	-	-
Unidentified spp.	8	6	6	6

[a] Percentage of total isolates based on 383 fungus isolates obtained from plating over 1,000 seeds.

One-pound samples of Ogden soybean seed were obtained from plots planted at biweekly intervals from April 15 to August 1 in 1952. In 1953, 1/4-pound samples of Roanoke, Jackson, and Lee were examined along with Ogden. Non-injured and injured seeds were plated to determine fungus association. These seeds were surface sterilized in mercuric chloride (1:1000)

[1] Plant Pathologist and Research Agronomist, respectively.

[2] Thanks are due Mr. Ralph Smith, Agronomist, Mobile Unit #2 of the North Florida Experiment Station, Quincy, Florida, for furnishing the seed samples used in these studies.

Table 2. Relative prevalence of fungi isolated from stink-bug punctured and non-punctured Ogden soybean seed obtained from plots planted August 1, 1952 and 1953, at Walnut Hill, Florida.

Fungus	Percent of total isolates[a]	
	Non-punctured seed	Punctured seed
Cercospora kikuchii	54	33
Chaetomium sp.	13	21
Fusarium oxysporum	6	10
Penicillium spp.	5	10
Aspergillus spp.	4	5
Cladosporium spp.	5	3
Helminthosporium spp.	4	1
Alternaria spp.	3	3
Aspergillus niger	3	Tr.
Phomopsis sojae	0	3
Fusarium roseum	0	1
Colletotrichum truncatum	0	1
Phyllosticta spp.	0	Tr.
Trichoderma sp.	0	Tr.
Colletotrichum destructivum	0	Tr.
Rhizopus nigricans	0	Tr.
Curvularia sp.	0	Tr.
Unidentified spp.	3	6

[a] Percentages based on fungi isolated from 300 non-punctured seeds and 1283 stinkbug-injured seeds plated on potato dextrose agar.

Table 3. Relative prevalence of stinkbug-injured Ogden soybean seed grown in a date-of-planting study at Walnut Hill, Florida in 1952-53.

Date of planting	Percentage of seeds injured[a]	
	1952	1953
April 15	2	22
May 1	3	14
May 15	5	25
June 1	7	25
June 15	4	49
July 1	9	72
July 15	-	76
August 1	45	82

[a] Percentages based on actual counts of 1 pound and 1/4 pound samples for 1952 and 1953, respectively.

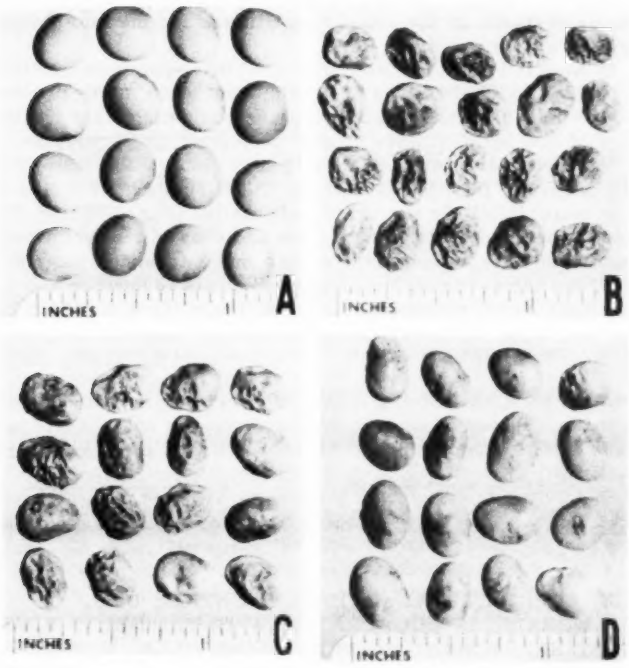

FIGURE 1. Soybean seed showing degree of southern green stinkbug injury on seed development; (A) Non-injured; (B), (C), and (D) showing severe, moderate, and light injury, respectively.

for one minute, washed in sterile, distilled water, and plated on potato dextrose agar containing 50 p.p.m. of the sodium salt of 2, 4-D acid[3]. The Petri plates containing five seeds each remained at room temperature (75° to 95° F.) for 8 days. The fungi growing from infected seeds were identified. The percentage of stinkbug-injured seed was determined by actual count of seed from all planting dates.

The relative amounts of the fungi obtained from stinkbug-injured seeds of different varieties are tabulated in Table 1. The percentage of seeds which yielded fungus growth when planted on agar were: Ogden 45, Jackson 42, Lee 36, and Roanoke 21. Cercospora kikuchii and Chaetomium sp. were most commonly isolated, while other fungi were less prevalent and all varied among varieties. Although C. kikuchii was the most frequent isolate, relatively few injured seeds showed visible purple staining on any of the four varieties prior to plating on agar.

Isolations were made fron stinkbug-punctured and non-punctured seed of the Ogden variety. The kind and percentages of fungi obtained are presented in Table 2. Thirty-seven percent of the non-punctured seed yielded fungus growth, while 34 percent of the punctured seed were fungus-infected after 8 days incubation on agar. The purple seed-stain fungus, C. kikuchii, was isolated more frequently from non-punctured seed than from stinkbug-injured seed. This suggests that stinkbug injury is not an important factor in the spread of purple seed stain. No satisfactory explanation can be given for the lower C. kikuchii infection of injured seed. Chaetomium sp., Fusarium oxysporum, and Penicillium spp. were isolated more frequently from the stinkbug-punctured seed, while other fungi were isolated only from such seed. Most of the latter fungi occurred sparingly, however, and the data suggest that stinkbug injury is not necessary for fungus infection of soybean seed.

The data for both years show that the percentage of stinkbug-injured seed was much higher at the later planting dates than at the earlier dates (Table 3). This trend was particularly evident in the 1953 season when stinkbug injury was much more prevalent than in 1952. The in-

[3]Hagborg, W. A. F., Gladys M. Warner and N. Agnes Phillips. 1950. Use of 2, 4-D as an inhibitor of germination in routine examination of beans for seed-borne infection. Science 111: 91.

crease in stinkbug injury in the later plantings may be largely attributed to migration of insects from a large area of earlier-maturing soybeans to the small area utilized for the late plantings. The four varieties were equally injured by stinkbugs.

Seed samples from the July 1 to August 1 planting dates indicated that symptoms of stinkbug injury to soybean seed varied, depending on the stage of development when injury occurred (Fig. 1). Some severely shriveled seeds had as many as 37 punctures on a single seed. Seeds severely punctured in an early stage of development were small, shriveled, and poorly developed (Fig. 1B). The punctures in the seed coats were sometimes very small when injury occurred at an early stage because of the failure of the seed to develop fully. Seeds injured at a later stage were not quite so distorted but were definitely shriveled and smaller (Fig. 1C) than non-injured seeds (Fig. 1A). Punctures in the seed coats were visible and showed a slight amount of discoloration surrounding the punctured area. Seeds injured in a more advanced stage of development were slightly shriveled, but the seed coat punctures were less in number and more noticeable. Each puncture was surrounded by a discolored ring 1 to 2 mm. in width (Fig. 1D). Such seeds were harmed much less than those punctured during an earlier stage of development.

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CURVULARIA LEAF BLIGHT OF RED CLOVER

K. W. Kreitlow and Helen Sherwin Yu[1]

Summary

Curvularia trifolii was identified as the causal agent of a leaf blight of red clover in Maryland and Florida. When isolates of the fungus from red clover were compared with isolates causing leaf blight of Ladino clover, they were identical, culturally, microscopically, and in pathogenicity.

A leaf blight of red clover not previously reported on this host in the United States has been observed since 1950, occasionally damaging red clover in Maryland and Florida. Curvularia trifolii (Kauff.) Boed., the causal agent of the blight, commonly attacks leaves of Ladino clover, Trifolium repens L., in pastures in the Eastern States. With the exception of a report from Japan, however, no records of the fungus attacking red clover, T. pratense L., have come to our attention. Occurrence of leaf blight of red clover in such widely separated areas as Maryland and Florida suggests that the disease is likely to occur elsewhere in the East on this host, but its presence may be obscured by the symptoms produced by other diseases.

Curvularia trifolii (formerly Brachysporium trifolii Kauff.) was first reported as causing minor damage to white clover in a lawn in the vicinity of Washington, D. C. (1). Since 1941, the fungus has been collected repeatedly on leaves of Ladino clover in the Eastern United States (3, 4). In 1930, Matsuura (5) reported the disease on red and white clovers as new for Japan. From results of artificial inoculation tests, however, Bonar (2) concluded that red clover was one of the least susceptible species among several clovers tested. There have been no other reports of the fungus attacking additional species of Trifolium.

Since 1950, five samples of leaves of red clover attacked by C. trifolii have been obtained. The first leaves damaged by the disease were collected from plots of red clover at Beltsville, Maryland, on October 30, 1950. At the time, it was thought that perhaps leaves of red clover were being damaged because of some unusual temperature and moisture relationships. However, leaves of Ladino clover also damaged by the fungus were collected at Beltsville, that same fall. Normally at Beltsville, leaves of Ladino clover are attacked by C. trifolii during periods of warm, humid weather in spring and summer; however, environmental conditions favorable for development of the disease sometimes occur in the fall. On October 18, 1951, leaves of red clover damaged by C. trifolii were again found at Beltsville, and on the same day the disease was found attacking Ladino clover in field plots nearby. The following spring (April 11, 1952), E. A. Hollowell collected specimens of diseased red clover at Fort Pierce, Florida. When the leaves were examined, numerous conidia of C. trifolii were found. On July 29, 1952, leaves of red clover attacked by C. trifolii were collected in field plots at Beltsville, Maryland. No symptoms of the disease were observed on red clover at Beltsville during the 1953 and 1954 growing seasons. However, E. A. Hollowell collected diseased leaves of red clover damaged by Curvularia on April 8, 1954, at Belle Glade, Florida.

Symptoms of the disease on red clover (Fig. 1) are similar to those produced by the fungus on Ladino clover. Lesions on leaves first develop as large, yellowed areas. These soon become watery gray, translucent and finally light brown. Leaves that are killed by the fungus turn brown to nearly black. When infection occurs near the base of a leaf, the fungus frequently attacks the petiole at the point where leaves are attached, causing the trifoliate leaves to collapse and droop characteristically. The symptoms resemble damage to red clover from attacks by the fungi causing northern anthracnose (Kabatiella caulivora (Kirchn.) Karak.) and southern anthracnose (Colletotrichum trifolii Bain & Essary). This suggests that attacks by Curvularia trifolii might be mistakenly attributed to the anthracnose fungi unless diseased leaves are examined microscopically. Curvularia leaf blight of red clover generally occurs in patches as though infection originated in a restricted area and spread outward. As many as 20 percent of the leaves within a given area were estimated to be damaged by the fungus.

When conidia of Curvularia trifolii from diseased leaves of Ladino and red clovers were

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[1] Senior Pathologist and Assistant Pathologist, respectively, Field Crops Research Branch, Agricultural Research Service, U. S. Department of Agriculture.

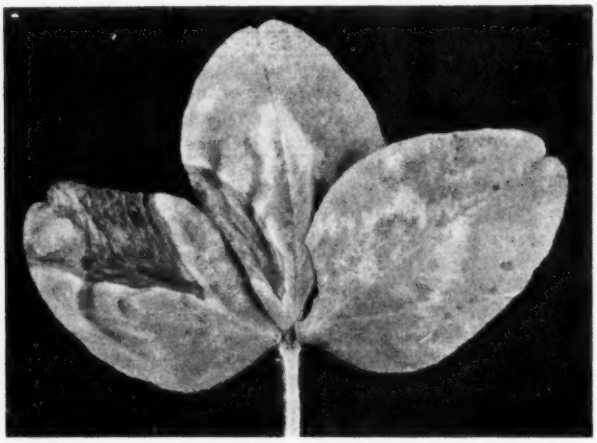

FIGURE 1.
Foliar symptoms of
Curvularia leaf blight
of red clover.

compared microscopically, they were similar in shape, size, septation, and color. Single-spore or multi-spore isolates of the fungus from red clover and Ladino clover grew similarly when cultured on potato-dextrose agar.

Pathogenicity tests were conducted in a greenhouse with cultures of Curvularia trifolii from both clover hosts. Three single-spore isolates from red clover and three from Ladino clover were grown for 20 days in Erlenmeyer flasks containing a mixture of wheat and oats. Inoculum of the three isolates from each host was pooled, dried and ground. A portion of the dried, pulverized inoculum from each of the clovers was then dusted on moistened leaves of six Ladino and six red clover plants growing in pots in a greenhouse. The inoculated plants were incubated in a moist chamber for 3 days and then transferred to a greenhouse bench at 70-80° F. Three uninoculated control plants of each host were incubated in the same manner.

Typical symptoms of the disease developed on leaves of inoculated plants of red and Ladino clover in 10 to 15 days. There was no difference in speed of infection or severity of symptoms regardless of isolates tested or hosts inoculated. The isolates of Curvularia trifolii from red clover attacked Ladino clover and the Ladino clover isolates attacked red clover with equal ease. No symptoms of disease developed on the check plants.

The results of these observations and tests suggest that the same strains of Curvularia trifolii that cause leaf blight of Ladino clover are the cause of a similar disease of red clover. The increasing prevalence of leaf blight on Ladino clover within the past 10 years may help to explain the occurrence of the disease on red clover. It is possible that Curvularia leaf blight of red clover may become more prevalent and destructive, particularly in the humid Eastern United States, and that it should be given consideration in developing disease-resistant varieties for this area.

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THE CLOVER ROOT BORER AS A VECTOR OF SOUTHERN AND NORTHERN ANTHRACNOSES OF RED CLOVER

F. W. Poos[1], J. L. Allison[2, 3], and K. W. Kreitlow[2, 4]

The close association of the clover root borer (Hylastinus obscurus (Marsh.)) with diseased plants of red clover (Trifolium pratense L.) seemed to be a strong indication that this insect could serve as an agent for disseminating and introducing disease-producing organisms into healthy plants. Exploratory tests were therefore conducted at Beltsville, Maryland, to learn more about this relationship.

Roots of red clover heavily infested with the root borer were obtained from R. T. Everly of Columbus, Ohio, in the spring of 1947. These roots were placed in a Berlese funnel in order to force adults to emerge as desired for use in the tests. Six pathogenic cultures of the southern anthracnose fungus (Colletotrichum trifolii Bain & Essary) were grown on potato-dextrose agar in petri plates until sporulation was abundant. On May 6 several borers were placed in each plate culture and allowed to crawl over the sporulating fungus growth for 10 minutes. Appendages from several selected root borers were examined microscopically and found to be heavily laden with conidia of the fungus. Five root borers from each culture were then transferred to the soil at the base of a single red clover plant. Plants used in this test had been grown in steam-sterilized soil in a greenhouse, cut back once, and repotted into 8-inch clay pots. Five additional root borers were transferred from one of the cultures to an additional red clover plant. A like number of root borers that had not been placed in contact with the cultures were transferred to each of three check plants. Each plant was enclosed in a cylindrical cellophane cage containing a wire-cloth ventilating window. The infested plants were kept in a greenhouse or insectary depending on which location more nearly approximated 60° to 70° F. All plants were examined periodically for macroscopic evidence of anthracnose infection, and within approximately 3 weeks typical symptoms were evident on all but one of the plants infested with the fungus-carrying borers. None of the check plants became infected. Colletotrichum trifolii was isolated from lesions on foliar parts of all plants on which the disease developed.

A second test started on May 13 produced only negative results, but the high temperature and low humidity during the period of this test were unfavorable for the development of southern anthracnose.

Further tests were initiated in 1952 when roots of red clover heavily infested with root borer were obtained from B. A. App of Columbus, Ohio. Tests as described above were repeated on April 25 and 28, except that four cultures of the northern anthracnose fungus (Kabatiella caulivora (Kirchn.) Karak.) as well as four cultures of the southern anthracnose fungus were used. Of 24 plants infested with the fungus-carrying root borers, 14 showed evidence of entance by this insect when examined on May 28. Eight plants were entered by borers carrying caulivora but only two plants developed symptoms of northern anthracnose. Six plants were entered by root borers carrying trifolii, but no plants developed positive symptoms of southern anthracnose. None of the check plants became infected. The low and somewhat inconsistent infection may be accounted for by the low humidity in the greenhouse during the period of the tests.

The results of preliminary studies reported here indicate that the clover root borer can be a vector of Colletotrichum trifolii and Kabatiella caulivora, the fungi which cause southern and northern anthracnoses of red clover, respectively. Its practical significance under field conditions needs to be determined.

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1, 2 Respectively, Entomology and Field Crops Research Branches, Agricultural Research Service, United States Department of Agriculture.

3 Present address: North Carolina State College, Raleigh.

4 The authors gratefully acknowledge the assistance of the late T. N. Dobbins in the 1947 tests.

OCCURRENCE OF A PLEOSPORA SPECIES ON CASTOR BEAN LEAVES

A. A. Cook

A species of Pleospora was found on leaves of Ricinus communis L. on three different occasions during the summer of 1954. Mature perithecia of this fungus were found twice on senescent leaves of the variety Cimarron growing at Gainesville, Florida, and once on senescent leaves of the variety U. S. 101 growing at Plymouth, North Carolina.

Perithecia were always well separated from one another, and never were more than 20 found in a single lesions. The lesions in which perithecia were found were light tan in color with distinctly defined borders but were usually not more than 5 mm. in diameter. The slightly embedded perithecia, which had short necks, measured 122-144 microns in diameter with ostiolar openings 37-43 microns in diameter. The ascospores, approximately 15 x 36 microns, each had 4-5 cross septae and 2-3 longitudinal septae (Fig. 1).

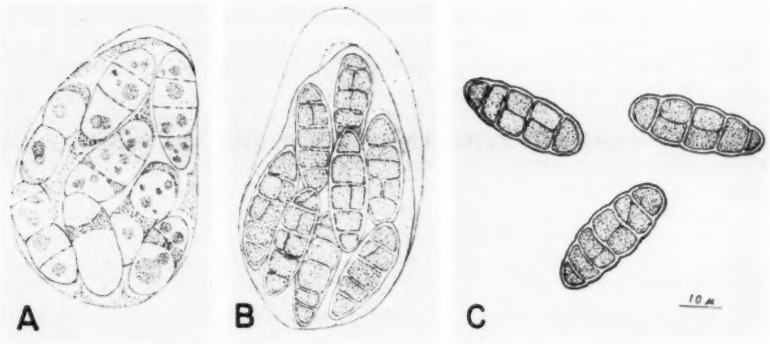


FIGURE 1. Stages of ascospore development of the Pleospora sp. found on senescent leaves of Ricinus communis; A, immature ascospores; B, nearly mature ascospores; C, mature ascospores.

Attempts to isolate single-ascospore cultures have been unsuccessful but mass hyphal tip cultures have been obtained. The organism grew rather slowly at room temperature on 2% potato-dextrose-agar, and although abundant perithecial initials were produced in culture, all have failed thus far to produce asci and ascospores. Inoculations from macerated hyphal cultures, with perithecia as described above, have been unsuccessful to date. Further study of this fungus, possibly a perfect stage of an Alternaria species, and its relation to a disease of castor bean is in progress.

FIELD CROPS RESEARCH BRANCH, AGRICULTURAL RESEARCH SERVICE, UNITED STATES DEPARTMENT OF AGRICULTURE, AND THE FLORIDA AGRICULTURAL EXPERIMENT STATION

UNUSUAL RECORDS OF PLANT DISEASE OCCURRENCE

OCCURRENCE OF YELLOW TUFT OF BENTGRASS IN RHODE ISLAND

By A. C. Tarjan and S. W. Hart

Yellow tuft was found during the fall of 1954 in a velvet bent turf experiment that had been initiated a decade ago to study the effect of cultural practices on the development of "thatch" (excessive organic matter accumulation). Although different rates of limestone and non-sterilized compost had been applied annually to this area, there seemed to be no correlation between rate or type of treatment and manifestation of yellow tuft symptoms.

Expressions of this disease appeared as described in earlier publications[1, 2]. Numerous small shoots were found arising from single stoloniferous nodes. Leaves making up these shoots were mostly yellow or necrotic.

It has been suggested that nematodes of the genera Panagrolaimus and Eucephalobus may, because of their activities in and around the growing point of a shoot, stimulate excessive shoot formation[2]. Examination of several tufts in the present study again revealed the presence of these genera as well as numerous nematodes of the genus Aphelenchoides in apparently healthy tissue. Other nematode genera observed were Aphelenchus and Rhabditis. These findings support the view that yellow tuft may be a group of concurrent symptoms in which the deleterious effects of nematodes play a part.

DEPARTMENT OF PLANT PATHOLOGY-ENTOMOLOGY AND AGRONOMY, RHODE ISLAND AGRICULTURAL EXPERIMENT STATION, KINGSTON

[1] Monteith, J., Jr., and A. S. Dahl, 1932. Turf diseases and their control. U. S. Golf Assn. Green Sect. Bul. 12 (4): 87-186.

[2] Tarjan, A. C., and M. H. Ferguson, 1951. Observation of nematodes in yellow tuft of bentgrass. U. S. Golf Assn. Jour. and Turf Management 4 (2): 28-30.

TOMATO NAILHEAD SPOT IN THE LOWER RIO GRANDE VALLEY

By G. H. Godfrey and A. L. Harrison

Tomato nailhead spot (Alternaria tomato) was found to a damaging extent in the Lower Rio Grande Valley of Texas in the fall of 1953. It occurred on stems as well as on green fruits. Infection varied from a single spot to a dozen or more spots per fruit. The spots were sometimes united, resulting in distortion of the fruit. Infection spread extensively in the affected areas after the first observation early in November. No fungicides had been applied at any time.

The last previous recorded observation on the occurrence of nailhead spot in this section of Texas was in November, 1947, when only a trace of it was seen. The origin of this unprecedented attack is not known. The outbreak followed the end of a long drought period when 7.56 inches of rain fell the last week in August, 0.98 inches in September, and 3.16 inches in October. There were frequent heavy dews at night throughout September, October, and November, with an occasional heavy fog lasting until 9 or 10 A.M. The temperature means were: in September 81.0°, in October 75.1°, and in November 66.4° F.

Accompanying the nailhead spot were light infections of early blight (Alternaria solani), moderately severe gray leaf spot (Stemphylium solani), and leaf mold (Cladosporium fulvum). The last-named increased to very severe late in the season.

Nailhead spot has been observed by the junior author only twice (1940 and 1949) in the Yoakum area of Texas. It likewise is considered to be of minor importance in the East Texas area (Jacksonville). Following the 1949 outbreak in the Yoakum area, where one field was a total loss, specimens were sent to Dr. G. F. Weber who verified the diagnosis as nailhead spot. Dr. Weber further stated that he had not seen the disease in Florida for a number of years, even though it was considered to be one of the major tomato diseases in Florida during the late 1920's and early 1930's. The causes of its sudden and erratic appearances in Texas have not been determined, but they recur frequently enough to constitute a warning that the Florida experiences of some 25 years ago could be repeated if favorable environmental conditions should again exist.

Addendum: Nailhead spot occurred again in the fall of 1954 in the Lower Rio Grande Valley of Texas. It was not quite so severe as in 1953, and was not accompanied by leaf mold. Rainfall has been much lower than last year, but heavy dews have occurred regularly. (G. H. Godfrey)

TEXAS AGRICULTURAL EXPERIMENT STATION, LOWER RIO GRANDE VALLEY EXPERIMENT STATION, WESLACO

CORRECTIONS

Regarding the article by B. A. Friedman, entitled "Association of internal brown spot of potato tubers with hot, dry weather" which appeared in the January 1955 number of the Plant Disease Reporter, please note the following corrections:

Sentence 2 of paragraph 4, page 39 should read "At the same time rainfall was well below normal as 8.49 inches fell as compared with the normal of 14.27 inches from June 15 to September 30."

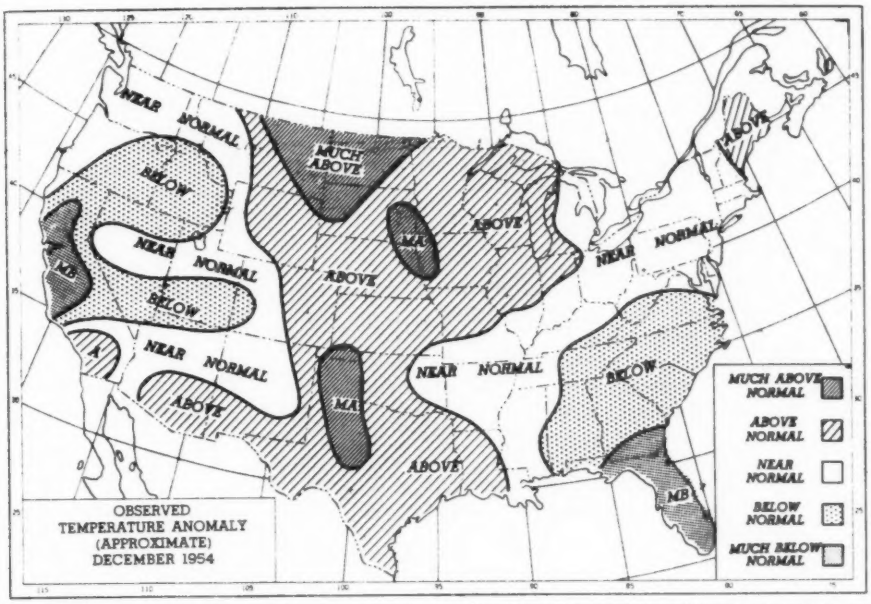
Reference 72 by Tabacs, on page 44, was published in 1930.

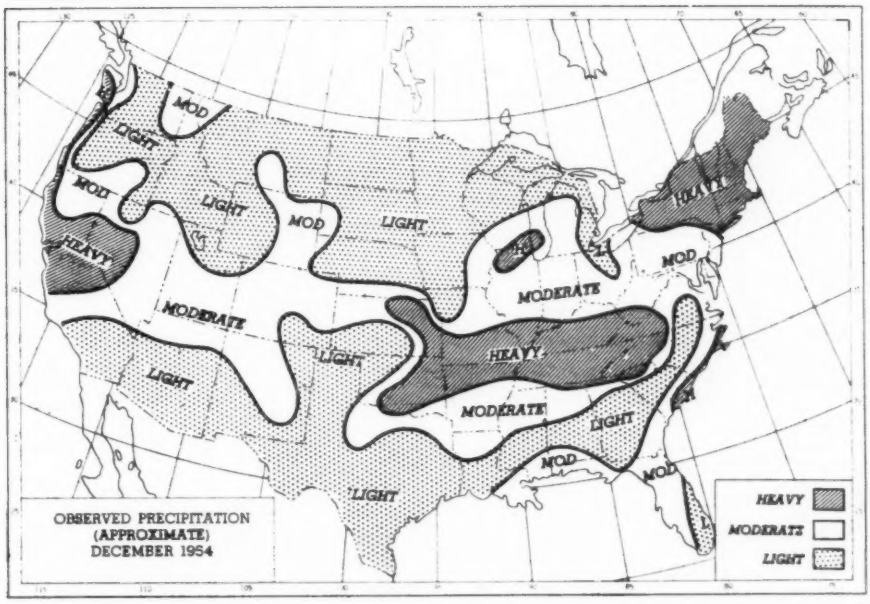
Also on page 44, the Biological Sciences Branch is in the Agricultural Marketing Service, not the Agricultural Research Service.

AMERICAN PHYTOPATHOLOGICAL SOCIETY
POTOMAC DIVISION MEETINGS SET FOR MARCH 3 AND 4, 1955

The two-day meetings of the Potomac Division of the American Phytopathological Society are set for March 3 and 4 at the Plant Industry Station, Beltsville, Maryland. Papers of general interest will be given in morning and afternoon sessions March 3, and a fungicide symposium, "Fungicides -- Past, Present, and Future", will be held the morning of March 4. This symposium will be moderated by Dr. J. W. Heuberger and the speakers are: Dr. George L. McNew, Dierector of the Boyce Thompson Institute for Plant Research, who will speak on the "History and Philosophy of the Use of Fungicides"; Dr. Hubert Martin, formerly of England, now Director of the Science Service Laboratory, London, Ontario, will speak on the topic, "The Mode of Action of Fungicides"; Dr. T. Walter Reed, Assistant Manager of Research, California Spray Chemical Corporation, with the topic, "Problems Encountered in the Commercial Production and Formulation of Fungicides"; and Mr. Lea S. Hitchner, Executive Secretary of the National Agricultural Chemicals Association, will discuss "Legal and Moral Aspects Encountered in the Use of Fungicides". Mr. Hitchner will place emphasis on the recently passed Miller Bill.

The speaker for the annual banquet the evening of March 3 will be Dr. Karl D. Butler. Dr. Butler is a trained plant pathologist with a number of years' experience as a pathologist in the State experiment stations in Arizona, Iowa, and New York, and as a pathologist in the tropics for the United States Department of Agriculture. Dr. Butler has become well acquainted with agricultural problems and with agricultural leaders throughout the United States in his capacity first as president of the American Institute of Cooperation, then as Executive Secretary of the National Institute of Animal Agriculture. He is also Farm Counselor to the Avco Manufacturing Corporation.. Dr. Butler"s topic will be "The Future of Agriculture."





The terms used in the accompanying maps, which define the ranges of temperature and precipitation, are numerical class limits. These are based on a statistical analysis of past records through which is determined the normal frequency of occurrence of temperatures and precipitation at various times of the year for different locations. For temperature the classes above, below, and near normal are so defined that they each normally occur one-fourth of the time; much above and much below normal, one-eighth of the time. Precipitation is depicted in terms of light, moderate, and heavy, each class normally occurring one-third of the time and thereby having equal probability of occurrence. These maps graphically represent only the general trends and give the country's weather picture at a glance. For quantitative studies, where monthly mean temperatures and actual precipitation records are needed for a given time and place, other publications of the Weather Bureau should be consulted. P. R. M.

MANUSCRIPTS FOR THE PLANT DISEASE REPORTER

(See also PDR 35: 121-122; 36: 214, 494)

(1) GENERAL: The Reporter page measures 9 inches long with the heading or 8 3/4 inches for the text part, by 6 inches wide. The copy is typed on a larger page, 11 1/4 inches of text or 12 inches overall in length by 8 inches in width, and reduced 25 percent in the photographic process of reproduction. Illustrations or tables larger in either dimension will take a correspondingly greater reduction. Only one size of type is available for text, footnotes, or tables.

(2) MANUSCRIPTS should be the original ribbon copy, not carbons, clearly typed and double-spaced throughout, including tables, footnotes, and bibliographies. (Note -- only one copy is needed.) Footnotes should be typed at the bottom of the page.

(3) ABSTRACTS are requested for all except very short articles. (PDR 38: 316).

(4) CAUSES OF DISEASES should be named. For bacteria, fungi, nematodes, etc., give the Latin name of the organism; for viruses either or both the accepted common name of the virus or a Latin name if you prefer it and there is one; for non-parasitic diseases state the causal factor if it is known. If the cause of a disease has not been determined say so. (PDR 36: 494).

(5) LITERATURE REFERENCES should be given in alphabetical order and numbered for citation in the text. We follow the AIBS suggestion of placing the year of publication after the author's name. Please check your titles carefully since we cannot do it for you. Be sure that text citations and bibliography agree; that foreign-language references are correct; that number or month is cited for periodicals that are not paged consecutively throughout the volume.

(6) NAMES OF FUNGICIDES should be given according to the suggestions that will be published in Phytopathology before long, and later repeated in the Reporter. In the meantime see PDR 35: 122.

(7) ILLUSTRATIONS should be sent to us unmounted. To prevent mistakes, write figure numbers on the back, and mark the top of each print when necessary. A sketch can show a preferred arrangement but please keep in mind page size, shape, and standard reduction (see above under General), and remember that figure titles and legends are part of the page. Lettering should be clear and large enough to be legible after reducing. Drawings, maps, and graphs can be photographs or originals, but should be finished and ready for reproduction, not just sketches.

(8) TABLES should be carefully thought out with particular attention to the Reporter's limitations in reproduction. Make titles and headings definite and self-explanatory. Designate footnotes in tables with superscript lower-case letters. Be sure that text discussion agrees with the data in the table. Do not abbreviate names of crop varieties.

(9) REPRINTS cannot be supplied since there is no way in which we can be reimbursed. However,

(10) The MULTILITH PLATES from which reprints can be made will be sent if requested at the time the article is submitted. The press size of these plates used for the Reporter is designated as small -- maximum image 9 1/2 by 13 inches, maximum paper size 9 3/4 by 14 inches -- for Model 1250. Most of the Experiment Stations have this type of multilith machine.

(11) We are the official correspondents in the United States of the PLANT PROTECTION BULLETIN published monthly by the United Nations Food and Agriculture Organization. We would appreciate it if plant pathologists would send us suitable material from time to time for inclusion in our monthly summary reports. Please mark it clearly so that we will know it is intended for the Bulletin. (See PDR 36: 492; 37: 637) -- P. R. M.

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